Version 0.7 Updated September 25th, 2006

Happy Happy Fun Fun

Ophthalmology textbook for the easily distracted

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CHAPTERS

Howdy partner!

This little pamphlet (I don't think it's big enough to call a book, yet) was written for a few express purposes:

1. Help you on your Step 2 boards
2. Inform you on basic ophthalmology concepts you might get asked in clinic.
3. Give me a chance to doodle (on paper, that is)

This text covers some basic topics and is definitely not "all inclusive." Unless you're "really into eyeballs" this material can be rather dry … so I've attempted to keep the chapters short, high-yield, and moderately entertaining. Obviously, I've left out large chunks of ophthalmology … for example, I didn't even mention uveitis (inflammation inside the eye). You should consult larger books for these topics if you're interested.

Famous Quotes

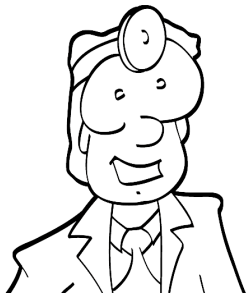
The reason why so few good books are written is that so few people who can write know anything.

Walter Bagehot

Pimping

The time-honored tradition of "pimping" began with Socrates and has trickled down through academia ever since. Each chapter ends with a series of "pimp questions" that might be asked during rounds. These questions also serve to reinforce the material and highlight important concepts for each chapter.

Finally, I'd like to mention that this book has never been "edited or peer reviewed." At the time of this writing, I am not a board-certified ophthalmologist, nor is this book endorsed by any residency program. This booklet is merely a collection of my thoughts that I am compiling for future puplication, and the content within should be taken with this in mind.

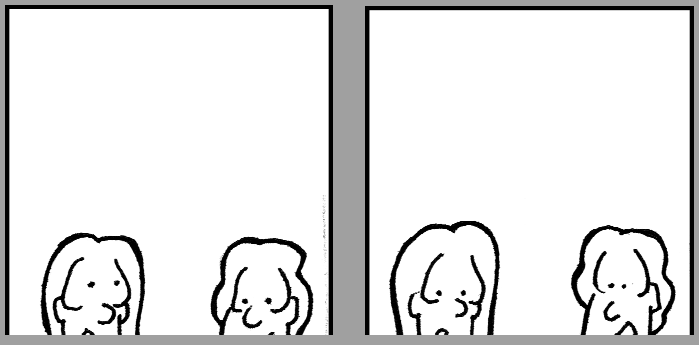


Thanks!

~Tim Root
www.timroot.com

Chapter One

Physical Exam



History and Physical for the Non-Ophthalmologist

by Tim Root
(last updated 9-25-06)

A thorough eye exam is important! Not only is the eye the most important organ in the body (of course!), but many common systemic diseases show ocular involvement. In fact, some conditions, like diabetes, may be first detected with the eye exam.

To help organize your eye exam, I've made a sample ophthalmology note on the facing page. Our clinic notes are difficult to interpret as our physical exam is long, specific, and requires many abbreviations to fit on a single page. Basically, it's a combination of a neurology and a dermatology note. Here's a rundown of the basic ophthalmology note's components.

History of Present Illness:

As with all other specialties, a detailed ocular history is crucial to diagnosis. You should explore every complaint with the "basic questions" -- **when** did it start, **what**'s it like, is there anything that makes it **better** or **worse**, are you taking any **medications** for relieve, etc.

Specific HPI review of systems should also include:

- **Floaters and flashing lights**: These are the classic symptoms of a retinal detachment and retinal tears so ask EVERY patient about these symptoms. Most patients complain of some floaters … see if they're actually new or have worsened recently.

- **Transient vision loss:** Think of migraine vessel spasm in the young and micro-emboli in the elderly. Curtains of darkness might indicate an ischemic event or a retinal detachment, so explore these symptoms in detail.

- **Blurry vision:** Is the vision always blurry? Does it worsen when reading or watching TV (people blink less when watching TV and develop dry eyes). Is this a glare problem at night that might indicate cataracts? Does the diabetic patient have poor control and hyperglycemic swelling of the lens?

- **Red, painful eyes:** A common complaint. Be sure to ask about the nature of the pain (is this a scratchy pain, aching pain, or only pain with bright light). Is there discharge that might indicate an infection?

- **Chronic itching and tearing:** Think about allergies or blepharitis. Is it in both eyes?

- **Headaches and scalp tenderness:** Think of temporal (Giant Cell) arteritis and ask about other collaborating symptoms like jaw claudication, polymyalgias, weight loss, and night sweats.

Sample Ophthalmology Note

HPI:

Example: 80 y.o. WF with history of NPDR (non-proliferative diabetic retinopathy) presenting with "blurry vision" in the right eye for the past 3 days. She said she was cleaning in her house, and "might have gotten something in my eye" two days ago. Since then she complains of darkness/blurryness of vision and photophobia. Some watering but no discharge OD. No flashes, possibly? new floaters (denies scalp tenderness, jaw claudication, polymyalgias, weight loss, night sweats, headaches, or any other systemic complaints). There has been no vision change in her left eye.

PMH :
HTN, CAD, DM2
(on oral agents)
asthma/COPD

POH
CE od in 2002
DES (dry eye syn)

FH
-Grandmother with glaucoma,
-no blindness in family

ALL
-PCN (rash)

Meds
- ATs
- "BP meds"
- Coumadin

Vcc *20/100 (PHNI)* / *20/20-1*

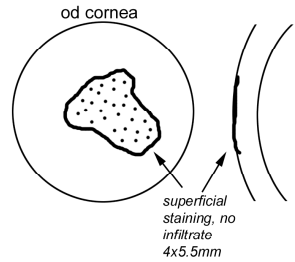
P *5 to 3* / *5 to 3* *(brisk, no apd ou)*

Tap *15* / *14*

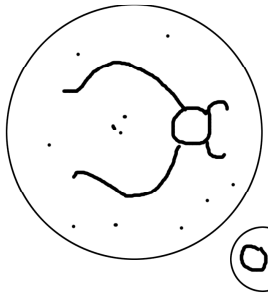
CF Full ou
EOMI ou

SLE

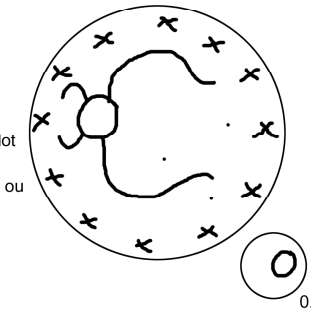
EXT : wnl ou
L/L : mild meibominitis ou
C/S : 1+inj. od, wnl os
K : see drawing
A/C : deep and quite ou
Iris : flat, round, no NVI ou
Lens : PCIOL od, 2+ NSC os
Vit : PVD ou, no cells ou



DFE



Macula: mild rpe atrophy OD
Vessels: arteriolar attenuation and AV nicking ou, no NVE
Periphery: PRP os, few dot-blot heme ou
Disk: CD ratio 0.3 ou, no NVD ou



A/P:

1. *Corneal Abrasion - appears sterile with no infiltrate. Possibly caused by scratch during house cleaning, vs. recurrant erosion, exacerbated by some lid lag. Will treat with emycin qid and qhs and have pt. tape lid at night. Will see daily until defect healed to insure it does not become infected.*
2. *NPDR - stable. Cont. to follow yearly and consider fundus photos*
3. *Return to oncall clinic tommorow*
4. *Pt. seen and discussed with attending*

~ Dr. Legible (pager 666)

Vcc (vision with glasses), **PHNI** (pinhole no improvement), **P** (pupils) **Tap** (pressure with applanation), **CF** (confrontational fields), **EOMI** (extraocular movements intact), **SLE** (slit lamp exam), **EXT** (external), **L/L** (lids & lacrimation), **C/S** (conjunctiva and sclera), **K** (cornea), **A/C** (anterior chamber), **Vit** (vitreous), **DFE** (dilated fundus exam), **CE** (cataract extraction), **LOL** (laugh out loud)

--- **OD** (right eye) **OS** (left eye) **OU** (both eyes) -----

The "Right Hand Column"

PMH:
Past medical history should include the basic health questions, but the main emphasis is on anything contributing to ocular problems -- such as diabetes, hypertension, and coronary artery disease. Also, ask about thyroid problems and asthma (you might need to prescribe a beta-blocker and you don't want to set off bronchospasm).

PMH :
HTN, CAD, DM2
(on oral agents)
asthma/COPD

POH
CE od in 2002
DES (dry eye syn)

FH
-Grandmother with glaucoma,
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-PCN (rash)

Meds
- ATs
- "BP meds"
- Coumadin

POH:
Ocular history should inquire about past ophtho visits and surgeries … specifically ask about cataract surgeries, eye trauma, and glaucoma. You can often piece together your patient's eye problems by examing their eyedrops.

Family History:
Focus on history of glaucoma and blindness. Patients will often confuse glaucoma with cataracts, so be sure to clarify this with them

Allergies:
List basic allergies and their reaction. We sometimes give Diamox to control eye pressure so make sure your glaucoma patient isn't allergic to sulfa drugs.

Meds:
Find out what eyedrops your patient is taking, and why. Are they using a regular eyedrop, or vasoconstricting Visine? Did they bring their drops with them? If your patient can't remember their eyedrops, it often helps to ask about the bottlecap-color of their eyedrops (ex. all dilating drops have red caps). Also, it's nice to know if your patient is taking an oral beta-blocker already.

Famous Quotes

Be careful about reading health books. You may die of a misprint.

Mark Twain

Vision, Pupil, and Pressure … oh my!

Vcc *20/100 (PHNI)* / *20/20-1* **P** *5 to 3* / *5 to 3* *(brisk, no apd ou)* **Tap** *15* / *14*

Vision, pupil, and pressure are the “vital signs” of ophthalmology. After a brief history, I check these measurements first BEFORE putting in dilating drops. If you ever consult ophthalmology, we will always ask you …

What’s the vision, pupil, and pressure?

It’s kind of a mantra … I don’t know how many times I’ve been told to “… get the vision, pupil, and pressure, then dilate them.” You see, dilating drops can negatively effect vision testing, pupil size, and elevate the pressure, so we need to check these parameters before dilation.

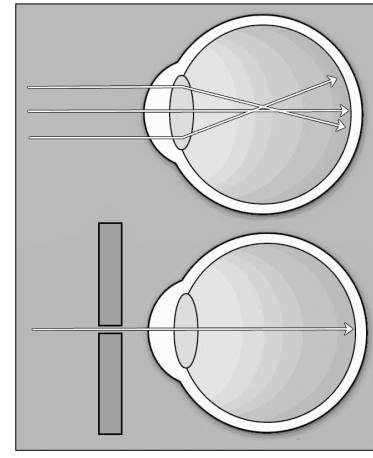
Visual Acuity:

You measure visual acuity with a standard Snellen letter chart (the chart with the BIG E on it. If your patient can’t read the E on the top line, see if they can count fingers at different distances. Failing this, try hand motion and light. Poor distance vision usually occurs from refractive errors (your patient needs better glasses).

You want to check a patient’s “best corrected vision” so have them wear their glasses. You’re going to be amazed at the number of people complaining of “blurry vision” who leave their glasses in their car. You’ll also be impressed by the number of consults you’ll get where the consulting doctor hasn’t checked the patient’s vision. Remember: “I can’t see!” is a relative complaint – for some this means 20/30 vision, and for others they “can’t see an atomic flash” (no light perception).

Das Pinhole!

A quick and easy way to determine whether refraction is the culprit, short of actually testing different lenses, is with the pinhole test. Punch a small hole in a paper card, and have your patient reread the eye-chart while looking through this pinhole. This can actually improve vision by several diopters. It works because the paper blocks most of the misaligned rays that cause visual blur, and allows the central rays to focus on the retina. If your patient shows no improvement with **pinholing**, start thinking about other visual impediments like cataracts or other media opacities. Our occluders (the black plastic eye cover) have a fold-down pinhole device for this purpose.



Near vision (or accommodation) can be assessed with a near-card or by having your patient read small print in a newspaper. Don’t try using the near-card to estimate distance acuity as distance vision is quite different than close-up acuity. That 20/20 marking printed on the near-card only checks “accomidated” near-vision. Remember that older patients can’t accommodate

well at near and need a plus-power lens (reading glasses) to help them read the card. Carry a +2.50 lens with you when seeing inpatients as 90% of these people leave their reading glasses at home.

Pupils:

The pupils should be equally round and symmetric with each other. You can test reactivity to light with a penlight, but a brighter light like the one on the indirect ophthalmoscope will work much better. When testing the eyes, you will see a **direct** constriction response in the illuminated eye, and a **consensual** response in the other eye. These should be equal and synchronous with each other.

Finally, be sure to check accommodation, the pupils should constrict with near-focusing.

The Swinging Light Test

If one eye is injured, or not sensing light, then your patient may have an APD or "afferent pupillary defect." Often the defect is only partial, making the APD difficult to detect on casual examination. To detect small APDs, you need to perform the "Swinging Light Test." Here's how it works . . .

When you shine a light back and forth between two normal eyes, you'll find that the pupils constrict, then dilate a fraction as the light beam passes over the nose, and then constricts again. As you go back and forth you'll see constriction, constriction, constriction, and constriction.

Things look different if one eye is partially blind. As before, when you shine the light in the good eye there is constriction. But, as you pass on to the other bad eye, both eyes seem to *dilate* a little. (The bad eye still senses light and constricts, but not as well.) So you see constriction, dilation, constriction, and dilation. This phenomenon is also called a "**Marcus Gunn pupil**."

Pressure:

We measure pressure by determining how much force it takes to flatten a portion of the corneal surface. There are several ways to do this … in the ophthalmology clinic we use the "Goldman Applanation Tonometer" that is attached to the slit-lamp.

In the ER, or with patients who are difficult to examine, we can check pressure using a handheld electronic Tono-pen. This little device can be tricky, and in the wrong hands becomes a random-numbers generator. I'll talk more about pressure in the glaucoma chapter.

Confrontational Fields:

All patients should have their visual fields (peripheral vision) checked. A patient may have great central vision (with perfect eye-chart scores), but suffer from "tunnel vision" resulting from neurological diseases or glaucoma. Your patient may not even be aware of this peripheral visual loss if it has progressed slowly over time.

Confrontational fields are easy to perform, but persuading your patient to concentrate may be tougher. Have your patient cover one eye, and tell them to look straight at your nose. While fixating on your nose, have them count your fingers as you flash them in different quadrants. Be sure to cover your own eye and hold your hands equidistant between you and the patient. This gives you a better idea of what your patient ought to be able to see … if you can see your fingers, your patient should be able to, as well.

EOMs (extraocular movements):

Check extraocular movements by having your patient follow your fingers into all quadrants. If the patient has decreased mobility in an eye from nerve paralysis or muscle entrapment, you may notice this from casual inspection or by more sophisticated cover/uncover tests. More often, though, you won't see anything but your patient will, complaining of double vision.

Seeing Double?

When evaluating double vision, you must first determine whether the doubling is *monocular* or *binocular*.

If, after covering an eye, the vision stays doubled, you know you're dealing with monocular diplopia. Monocular diplopia isn't a neurological problem, but likely from a refractive error such as astigmatism, cataract, or corneal surface wrinkling.

Binocular diplopia indicates a misalignment between the eyes … and this is likely due to neuromuscular paralysis or muscle entrapment (if after trauma). To tease out what muscle groups and nerves are involved, you should determine what gaze direction improves and worsens the doubling. We'll discuss this in more detail in the neuro chapter.

The slit-lamp Exam:

It takes several months to get good at using the slit-lamp microscope, and it doesn't help that we use several different types of microscope in our clinic. We describe our findings in the same order with every patient … starting from the outside skin and working our way to the back of the eye.

SLE

EXT : wnl ou
L/L : mild meibominitis ou
C/S : 1+inj. od, wnl os
K : see drawing
A/C : deep and quite ou
Iris : flat, round, no NVI ou
Lens : PCIOL od, 2+ NSC os
Vit : PVD ou, no cells ou

External Exam (EXT):
When examining the external eyes, make sure the eyes look symmetrical and that the patient doesn't exhibit ptosis (drooping of the eye) or proptosis (extruding eyes or "bug-eyes"). Check to make sure the sclera is white and non-icteric, and the conjunctiva blood vessels aren't injected (red and inflamed). If the patient has a conjunctivitis, check for a swelling of the pre-auricular nodes (in front of the ear) and sub-mandibular/mental nodes.

Lids and lacrimation (L/L):
Always look at the lid margin and lashes for signs of blepharitis. Evert the lids to look for follicles or papillary bumps that might indicate infection or irritation.

Conjunctiva and Sclera (C/S):
See if the conjunctival blood vessels look injected. If they are injected, do they blanch white when you dilate the patient with phenylephrine?

Cornea (K):
Look at the corneal surface for erosions and abrasions that might indicate drying or trauma. Does the stroma look clear? Look at the back endothelial surface for folds or gutatta bumps.
Fluorescein dye will make surface abrasions easier to spot.

Anterior Chamber (AC):
Look for cell and flare, which could indicate inflammation or a bleed. Individual cells are hard to see … you need to turn the lights down low and shoot a "ray of light" into the eye. If you compare this light to a projector beam at the movies, then "cell" will look like dust flecks while "flare" is diffuse and looks like smoke. Also, comment if the anterior chamber is deep and well-formed, or shallow (a setup for angle-occlusion glaucoma).

Iris (I):
Make sure the iris is flat and the pupil round. If the patient has diabetes or an old retinal vascular occlusion, comment whether you see any signs of neovascularization of the iris.

Lens (L):
Is the lens clear, or hazy with cataract. Are they phakic (their own lens), pseudophakic (prosthetic lens), or aphakic (no lens at all)?

Vitreous (V):
You can look behind the lens into the dark vitreous cavity. If you suspect a retinal hemorrhage or detachment, you may see cells floating here.

Fundus Exam:

The fundus is the only place in the body where you can directly visualize blood vessels and nerves. In our notes we typically comment on four retinal findings:

Macula – is it flat. Is there any RPE atrophy?
Vessels – Any signs of AV nicking? Attenuation of the arterioles?
Periphery – Any lattice, cobble-stoning, tears?
Disk – Cup to disk ratio. Do the rims look pink? Papilledema?

You're probably not going to get good at the retina exam at first, but do your best.

The Direct ophthalmoscope

For non-ophthalmologists, the most common way to examine the fundus is with the direct ophthalmoscope. This hand-held device is not easy to use, however, especially in an undilated eye. The key to success is to get the instrument and yourself as CLOSE to the patient as possible. Get really close! Dilating the eye also helps.

Using that darn direct scope

Switch the light to the highest setting, and rotate the beam to the medium-sized round light. I set my focus ring to "0," but you may need to adjust this to compensate for your own refractive error. Place your hand on your patient's shoulder or head.

Starting far away, find your patient's red-reflex and follow that reflection in as you close in to the eye. Be sure to switch eyes so that you don't end up face-to-face with the patient (unless they are extremely attractive).

It may take you a while to visualize the fundus with this direct scope, especially in undilated eyes, because the field-of-view you get is very small, making it hard to even recognize what you are seeing.

I find it easiest to find a blood vessel and then follow this vessel back to its origin at the optic disk. Inspect the disk margins and the size of the disk cupping. You may be able to pick up AV nicking from high blood pressure and retinal hemorrhages in the form of dot blot spots or flame hemorrhaging.

At the slit-lamp

The best way to look at the posterior fundus in magnified detail is with a lens at the slit-lamp. This is how we look at the optic nerve and macula in the clinic, but it takes practice.

The Indirect Ophthalmoscope

This is how we look at the peripheral retina in the ophtho clinic. The eye needs to be dilated to get a good view … but the field of view is excellent.

Other Tests Specific to Ophthalmology:

There are a couple of other exam techniques specific to ophthalmology, so you probably won't be exposed to them unless you go into the field … like gonioscopy to look at the iris-corneal angle by using mirrored lens. I'll cover these topics in later chapters as we come to them.

1. What are the three "vital signs of ophthalmology" that you measure with every patient?

Vision, pupil, and pressure. Some attendings might say there are five vital signs (including extraocular movements and confrontational fields.)

2. What is a Marcus Gunn pupil?

Simply an APD (afferent papillary defect)

3. How do you perform the Swinging light test?

You shine a light back and forth between the pupils. You should see "constriction-constriction-constriction-constriction" as you flip-flop. If you see constriction-dilation-constriction-dilation … then something's wrong. You have an APD.

4. What is "pinholing" a patient?

Using a pinhole to correct some of their refractive error. When patients significantly improve with pinholing, they probably need new glasses.

5. When presented with a complaint of "double vision" what is the first thing you should determine?
Whether the double vision is binocular or monocular … as this completely changes your differential. Monocular diplopia is a refractive error, while binocular diplopia is a misalignment between the eyes (and a major headache to figure out the cause).

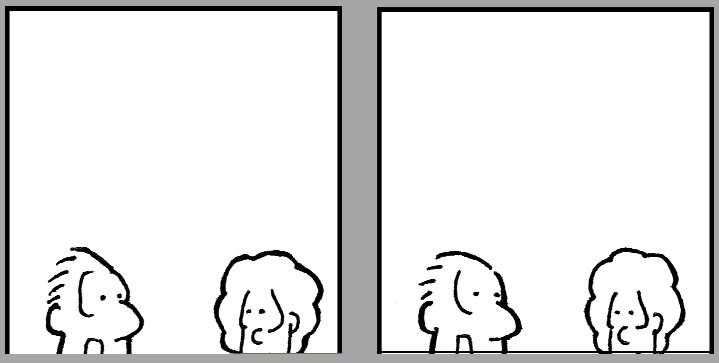
6. What is cell and flare?
These are descriptive terms to describe inflammation in the anterior chamber. Flare is protein floating in the aqueous that looks like a projector beam running through a smoky room. Cells are individual cells that look like dust-specks floating through that same projector beam.

7. You are thinking of starting eyedrops to control the eye pressure in a newly diagnosed glaucoma patient. What medical conditions might you ask about before initialing therapy?

Eyedrops can have pretty impressive systemic side effects as they bypass liver metabolism and absorb through the nasal mucosa. Be sure to ask about heart problems and asthma before starting a beta-blocker.

Chapter Two

Eye Anatomy

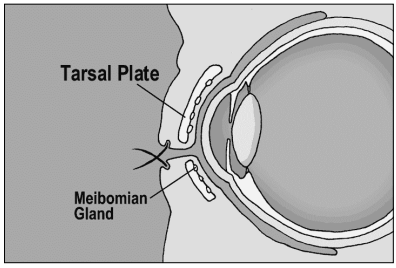


Basic Eye Anatomy

by Tim Root
Updated 7-3-06

Before discussing conditions affecting the eye, we need to review some eye anatomy. Anatomy can be painful (I hated anatomy in med school) so I'm going to keep this simple. Let us start from the outside and work our way inwards.

Eyelids



The eyelids protect and help lubricate the eyes. The eyelid skin is very thin, containing no subcutaneous fat, and is supported by a **tarsal plate**. This tarsal plate is a fibrous layer that gives the lids shape, strength, and a place for muscles to attach.

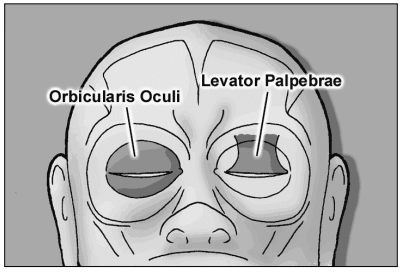
Underneath the tarsal plate lie **meibomian glands**. These glands secrete a lipid substance into the tear film that keeps the tears from evaporating. Meibomian glands may become inflamed, swelling into a **chalazion** that needs to be excised. Don't confuse a chalazion with a stye. A **stye** is a pimple-like infection of a sebaceous gland or eyelash follicle, similar to a pimple, and is superficial to the tarsal plate. Styes are painful, while chalazions are not.

Famous Quotes

You can't depend on your eyes when your imagination is out of focus.

Mark Twain

Eyelid Movement

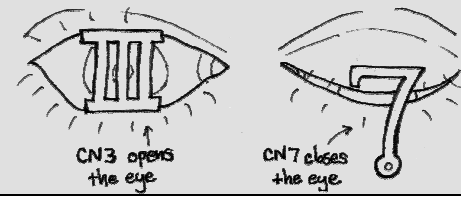


Two muscles are responsible for eyelid movement and they both attach to the tarsal plate. The **orbicularis oculi** closes the eyelids, and it is innervated by cranial nerve 7. Patients with a facial nerve paralyses, such as Bell's Palsy, can't close their eye and the eye may need to be patched (or sutured closed) to protect the cornea. The **levator palpebrae** opens the eye. It is innervated by CN3, and oculomotor nerve palsy is the major

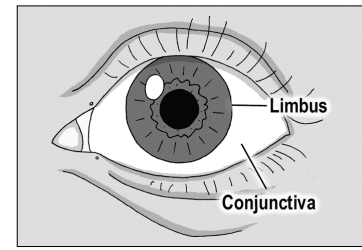
cause of ptosis (drooping of the eye). In fact, a common surgical treatment for ptosis involves shortening the levator to open up the eye.

Mnemonic

Cranial 3 opens the eye like a pillar
CN 7 closes like a fish-hook



Conjunctiva

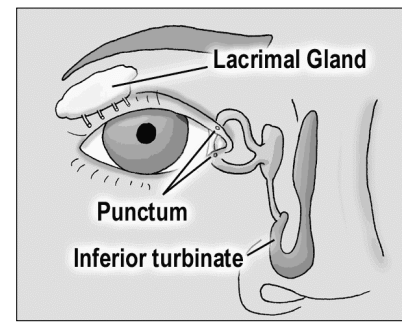


The conjunctiva is a mucus membrane that covers the front of the eyeball. When you examine the "white part" of a patient's eyes, you're actually looking through the semi-transparent conjunctiva to the white sclera underneath. The conjunctiva starts at the edge of the cornea (this location is called the **limbus**). It then flows back behind the eye, loops forward, and forms the inside surface of the eyelids. The continuity of this conjunctiva is important, as it keeps objects like eyelashes and your contact lens from sliding back behind your eyeball. The conjunctiva is also lax enough to allow your eyes to freely move. When people get conjunctivitis, or "pink eye," this is the tissue layer affected.

Fun Fact!

There is a thickened fold of bulbar of conjunctiva called the semilunar fold that is located at the inner canthus … it is a homolog of the nictitating membrane seen on sharks.

Tear Production and Drainage

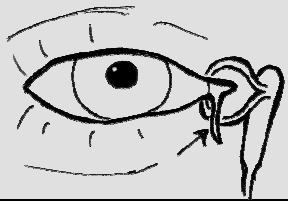


The tears are produced by glands in the lid and conjunctiva, and also by the lacrimal glands. Lacrimal tears flow down the front of the eye and drain out small pores, called **lacrimal punctum**, which arise on the medial lids. These puncta are small, but can be seen with the naked eye.

After entering the puncta, tears flow down the lacrimal tubing and eventually drain into the nose at the inferior turbinate. This explains why you get a runny nose when you cry. In 2-5% of newborns, the

drainage valve within the nose doesn't open. This often resolves on it's own, but sometimes we need to force open the pathway with a probe.

Lid Lacerations
Most lacerations through the eyelid can be easily reopposed and repaired. However, if the laceration occurs in the nasal quadrant of the lid, you have to worry about messing up the tear-drainage pathway … that's when you definitely want ophtho to evaluate for lacrimal damage.

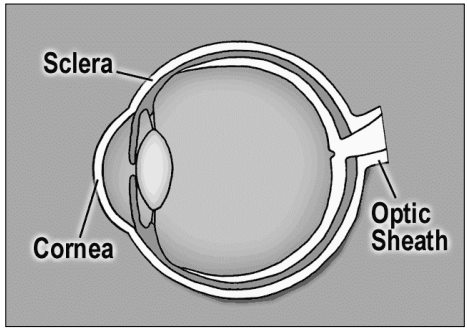


Warning: Drug absorption through the nasal mucosa can be profound as this is a direct route to the circulatory system and entirely skips liver metabolism. Eyedrops meant for local effect, such as beta-blockers, can have impressive systemic side effects. Patients can decrease nasal drainage by squeezing the medial canthus after putting in eyedrops. They should also close their eyes for a few minutes afterwards because blinking acts as a tear pumping mechanism.

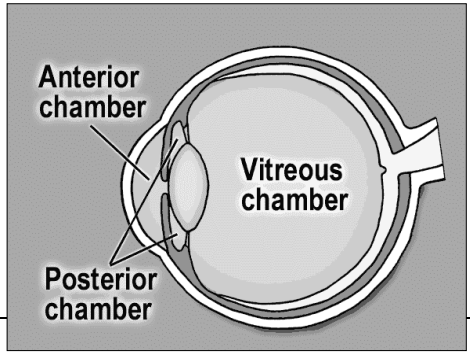
The Eyeball:

The eyeball is an amazing structure. It is only one inch in diameter … roughly the size of a ping-pong ball, and is a direct extension of the brain. The optic nerve is the only nerve in the body that we can actually see (using our ophthalmoscope, that is).

The shape of the eye is created by the **sclera**. The sclera is white, fibrous, composed of collagen, and is actually continuous with the clear cornea. In fact, you can think of the cornea as an extension of the sclera as they look similar under the microscope. The cornea is clear, however, because it is relatively dehydrated. At the back of the eye, the sclera forms the optic sheath encircling the optic nerve.

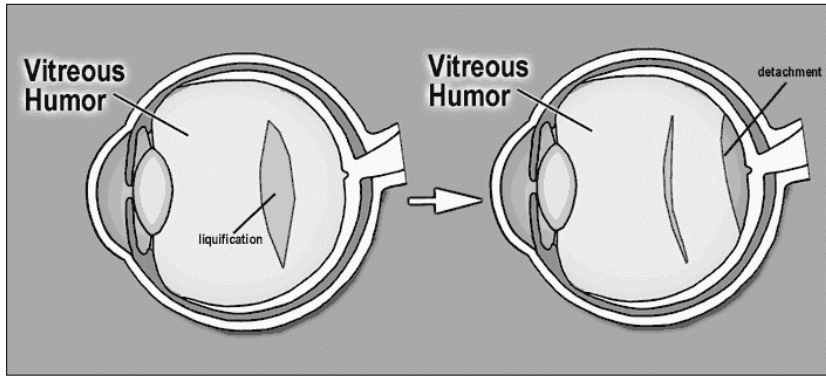


The eyeball is divided into three chambers (not two, as you might expect). The **anterior chamber** lies between the cornea and the iris, the **posterior chamber** between the iris and the lens, and the **vitreous chamber** lies behind the lens going back to the retina.



The eye is filled with two different fluids. **Vitreous humor** fills the back

vitreous chamber. It is a gel-like suspension with a consistency similar to Jell-O. Not only does the vitreous help maintain the eye's shape, but it also helps refract light. The mature eye no longer produces this vitreous humor, therefore, when it is lost through trauma or surgery, we have to replace it with saline or aqueous. With age and certain conditions, areas of the vitreous can liquefy. If this occurs, the vitreous can fall in upon itself – this is usually a harmless event called a PVD (posterior vitreous detachment). However, this vitreous detachment can pull on the retina and create a retinal detachment. A vitrectomy is a surgery where the vitreous is removed.



Aqueous humor fills the posterior and anterior chambers. It is a watery solution, with a high nutrient component. It is continuously produced in the posterior chamber, flowing forward through the pupil into the anterior chamber, where it drains back into the blood circulation via the Canal of Schlemm. Its content is similar to plasma, however it has much higher levels of lactate. We'll discuss the aqueous pathway in detail in the glaucoma chapter.

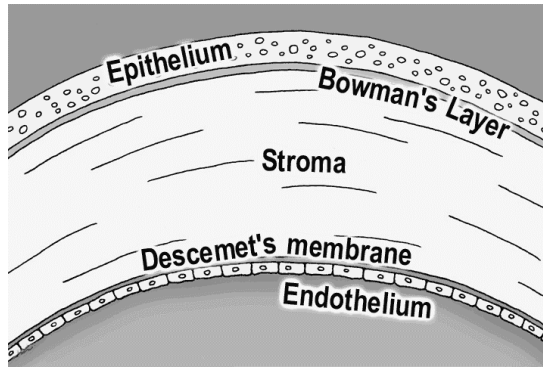
Famous Quotes

You can lead a horse to water, but you can't make him float.

Unknown

The Cornea:

The **cornea** is the clear front surface of the eye. The cornea-air interface actually provides the majority of the eye's refractive power. The cornea is avascular and gets its nutrition from tears on the outside, aqueous fluid on the inside, and from blood vessels located at the periphery.



On cross section, the cornea contains five distinct layers. The outside surface layer is composed of epithelial cells. This is where patients will get corneal abrasions. Though an epithelial injury is painful, this layer heals very quickly and does not scar. Under this lies Bowman's layer and then the stroma. The corneal stroma makes up 90% of the corneal thickness, and if the stroma is damaged, it *can* lead to scar formation. The next layer is Descemet's membrane, which is really the basal lamina of the endothelium, the final layer.

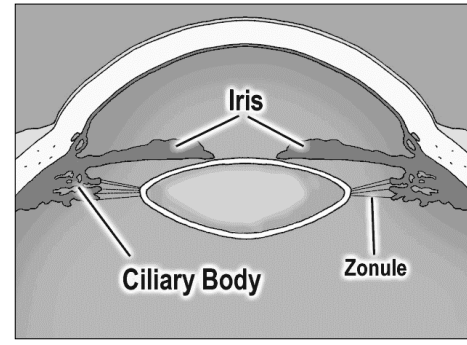
The endothelium is only one cell layer thick and works as a pump to keep the cornea dehydrated. If the endothelium becomes damaged (during surgery or by disease processes) aqueous fluid can flow unhindered into the stroma and cloud up the cornea with edema. Endothelial function is very important as these cells don't regenerate when destroyed – the surviving endothelial cells just get bigger and spread out. If the cell count gets too low, then the endothelial pump can't keep up and the cornea swells with water … necessitating a corneal transplant to regain vision.

Mnemonic

Decemet's membrane is "deep," while Bowman's layer is high up in the "belfry." A belfry is a room, usually high up in a tower, where bells are hung.

The Anterior Chamber Angle:

The angle formed by the inner cornea and the root of the iris is particularly important in ophthalmology. Here you find the trabecular meshwork with its underlying Schlemms canal. This is where aqueous is drained … and blockage of this pathway/angle will become important as we discuss glaucoma.

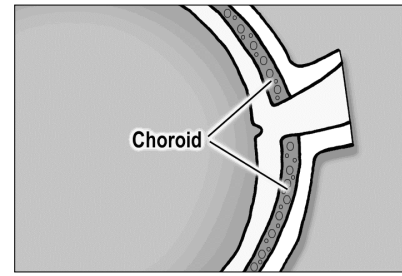


The Uvea:

The iris, ciliary body, and the choroid plexus are all continuous with each other and are collectively called the **uvea**. This is an important term, as many people can present with painful "uveitis" - spontaneously or in associated with rheumatologic diseases.

The **iris** is the colored part of the eye and its primary function is to control the amount of light hitting the retina. Sympathetic stimulation of the pupil leads to pupil dilation and parasympathetic stimulation leads to constriction. In other words… if you see a bear in the woods, your sympathetics kick in, and your eyes dilate so you can see as much as possible as you run away.

The inner iris flows back and becomes the **ciliary body**. The ciliary body has a couple of functions. It creates the aqueous fluid that fills the posterior and anterior chambers. Additionally, the ciliary body has muscles that change the lens shape by relaxing the zonular fibers that tether to the lens capsule.



The **choroid** is a bed of blood vessels that lie right under the retina. The choroid supplies blood to the outer one-third of the retina, including the actual rod and cone photoreceptors. This blood is delivered by diffusion. Retinal detachments occur between the retina and the choroid, which is disastrous for the photoreceptors as they quickly die without this choroidal nourishment.

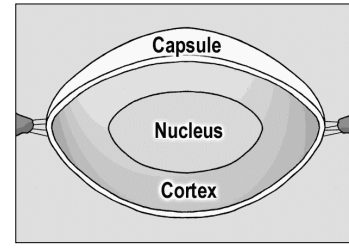
Fun Fact!

An ostrich's eye is larger than its brain.

Lens:

The lens sits behind the iris. The lens is kinda neat because it doesn't have any innervation or vascularization. It gets its nourishment entirely from nutrients floating in the aqueous. It also has the highest protein concentration of any tissue in the body (65% water, 35% protein).

The lens itself is made of three layers … in a configuration similar to a peanut M&M. The outer layer is called the **capsule**. The capsule is a thin layer, with a consistency of saran wrap, which holds the rest of the lens in place. The middle layer is called the **cortex**, while the central layer is the hard **nucleus**. Cataracts are described by where they occur



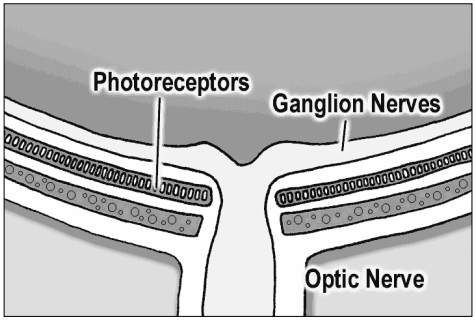
… such as nuclear cataracts, cortical cataracts, and subcapsular cataracts. With cataract extractions, the capsule is left behind, and the artificial lens is placed inside this outer bag.

The capsule is held in place by suspensory ligaments called "zonules" or "the zonules of Zinn" that insert around the periphery of the capsule and run to the muscular ciliary body. Contraction of the ciliary muscle causes the zonule ligaments to *relax* (think about that for a minute), allowing the lens to become rounder and increase its refracting power for close-up reading.

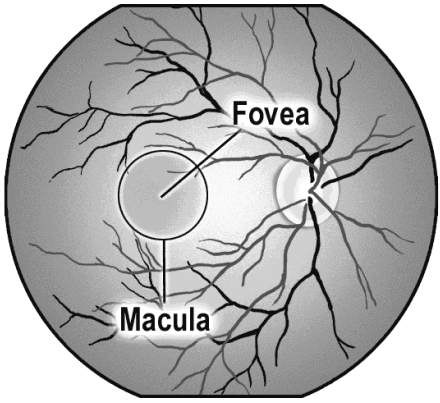
As our lens harden with age, the lens becomes less pliable. The ciliary body isn't strong enough to warp the stiff lens so we lose our ability to read close-up. In children the lens is soft like squishy plastic, but as you age it hardens to glass. In older people, the ciliary body contracts, but the lens is unable to relax and "round out," thus leading to problems accommodating (focusing on near objects like newspapers). This is called presbyopia. Almost all older people need reading glasses because of hardening of their lenses.

The Retina:

The **retina** is the sensory portion of the eye and contains layers of photoreceptors, nerves, and supporting cells. Histologically, many cell layers can be seen, but it is probably not worthwhile to memorize them at this point. The important ones include the photoreceptor layer, which is located further out (towards the periphery), and the ganglion nerve layer which lies most inward. For light to reach the photoreceptor it has to pass through many layers. After light reaches the photoreceptor rods and cones the visual signal eventually propagates to the ganglion nerves. These ganglion nerves, in turn, course toward the optic disk and form the optic nerve running to the brain.



The **macula** is the pigmented area of the retina that is responsible for central vision. Within the central macula lies the **fovea**, which is a small pit that is involved with extreme central vision. The fovea is very thin and derives its nutrition entirely from choroidal diffusion, making it even more susceptible to injury during retinal detachments.



The optic disk is the entry and exit point of the eye. The central retinal artery and

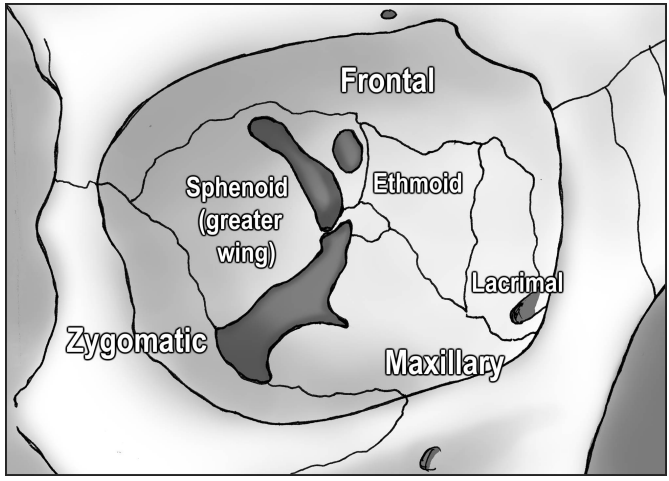
vein pass through here, as do the ganglion nerves as they form the optic nerve.

The Orbital Walls:

Seven different bones compose the orbit … don't be intimidated by this complexity, however, as it's not that confusing. For example, the roof of the orbit is a continuation of the frontal bone, the zygomatic bone forms the strong lateral wall, while the maxillary bone creates the orbital floor. This makes sense, and you could probably guess these bones from the surrounding anatomy.

The medial wall is a little more complex, however, but is mainly formed by the lacrimal bone (the lacrimal sac drains tears through this bone into the nose) and the ethmoid bone. The thinnest area in the orbit is a part of the ethmoid bone called the **lamina papyracea**. Sinus infections can erode through this "paper-thin wall" into the orbital cavity and create a dangerous orbital cellulites.

Despite the fragility of the medial wall, it's the orbital floor that most often breaks during blunt trauma. The maxillary bone fractures downward and the orbital contents herniate down into the underlying maxillary sinus. This is called a "blowout fracture" and can present with enopthalmia (a sunken-in eyeball) and problems with eye-movements from entrapment of the inferior rectus muscle. We'll discuss blow-out fractures in more detail in the trauma chapter.



The back of the orbit is formed by the sphenoid bone, with the "lesser wing" of the sphenoid forming around the optic canal itself. There's also a little palatine bone back there in the middle, but don't worry about that one!

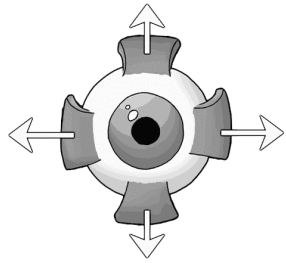
The Apex: Entrance into the Orbit

The Orbital Apex is the entry point for all the nerves and vessels of the eye. The superior orbital fissure lies between the wings of the sphenoid bones, through which many veins and nerves pass into the orbit. Among these nerves are the frontal and lacrimal nerves (branches of V1).

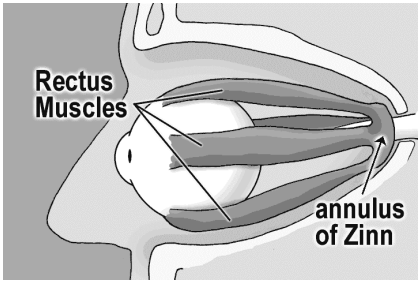
The "Annulus of Zinn," a muscular band that serves as the insertion point for most of the ocular muscles, rests on top of the superior orbital fissure. Also within the Annulus of Zinn, are the optic nerve and ophthalmic artery (the major blood supply to the orbit) which pass through the optic canal.

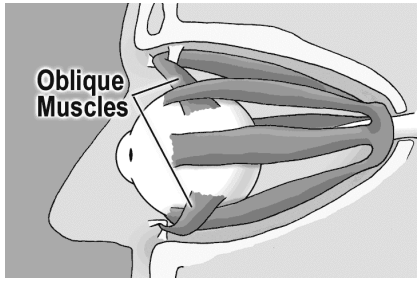
Eye Muscles:

Four **rectus muscles** control each eye. These muscles insert at the sclera, behind the limbus, and each pull the eye in the direction of their attachment.



The superior, medial, and inferior rectus muscles are all controlled by the oculomotor nerve (III). The lateral rectus, however, is controlled by the abducens (VI) nerve, which makes sense, as the lateral rectus abducts the eye.





The remaining two eye muscles are the superior and inferior **oblique muscles**. These muscles also insert in the posterior orbit, but course nasally until they reach trochlea (or "pulleys") before inserting onto the eye. Because of this insertion, the oblique muscles are primarily responsible for intorsion and extorsion (rotation of the eye sideways), though they also contribute to gaze direction.

We will talk more about these muscles and their innervation in other chapters, when we discuss neurology.

Blood Supply and Drainage:

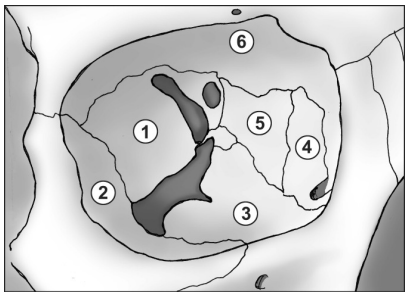
Ophthalmic artery
The ophthalmic artery is the first branch off the internal carotid artery, and is the main blood supply to the orbit. The first infraorbital branch off the ophthalmic is the central retinal artery which accompanies the optic nerve through the optic canal. Other branches supply the orbital muscles, lacrimal gland, etc..

Venous Drainage:
Through the superior and inferior ophthalmic veins which communicate to the cavernous sinus. There is a direct communication of the venous drainage from the skin of the periorbital skin to the cavernous sinus - an infection of the periorbital skin can lead to a deadly cavernous sinus thrombosis!

Summary:
There is obviously much more anatomy you can learn, but we should probably hold off for now and discuss further details in future chapters as they become relevant.

1. Why don't objects like contact lens and eyelashes get stuck behind the eye?
Because the conjunctiva covering the front of the eye loops forward and covers the inside of the eyelids as well.

2. How many chambers are there in the eyeball?
Three, actually. There is the anterior chamber in front of the iris, the posterior chamber between the iris and the lens, and the vitreous chamber behind the lens which fills most of the eye.



3. Name each of the numbered bones.

… which bone is thinnest?

… which is most likely to fracture after blunt injury?

… which is most likely to erode from sinus infections?

The bones are: (1)Sphenoid (2)Zygomatic (3)Maxilla (4)Lacrimal (5)Ethmoid (6)Frontal. The ethmoid is the thinnest bone and most likely to perforate from an eroding sinus infection (happens mostly in kids). The maxillary floor is most likely to fracture from blunt injury.

3. How could an infection track back to the cavernous sinus?
Remember the "danger triangle?" That's the area from the bridge of the nose down to corners of the mouth. Infections in this area can track up the superior ophthalmic veins into the cavernous sinus and beyond … and cause problems.

4. What is the uvea? What eye structures compose it?
The uvea comprises the iris, ciliary body, and the choroid. They are all connected to each other and are histologically similar. Patients can present with a painful "uveitis," usually secondary to rheumatological conditions like sarcoidosis.

5. Where does the retina get its nutrition supply?
The inner 2/3rds of the retina (inner implies toward the center of the eyeball) gets it's blood from the retinal vessels. The outer 1/3 (which includes the rods and cones photoreceptors) gets it from the choroid plexus. A retinal detachment, which separates the retina from the choroid, is particularly dangerous for the photoreceptors, especially if the detachment involves the macula (as the macula gets its blood supply primarily from the underlying choroid).

6. When the ciliary body contracts, how does the lens change shape (does it get rounder or flatter)?
When the ciliary body contracts, the zonules actually *relax* and the lens relaxes and gets rounder. With age, the lens hardens and has a hard time relaxing, no matter how hard the ciliary contracts. This aging process is called presbyopia.

7. What eye muscle attaches right behind the macula?
The inferior oblique attaches here. This is very important when performing inferior oblique muscle surgery.

8. Which full-thickness eyelid laceration is more dangerous … medial or lateral lacerations? Why??
You worry about the canalicular tear-drainage system involvement with medial lacerations.

9. How many layers are there in the cornea … can you name them?
The superficial Epithelium, Bowman's layer, Stroma, Decemet's membrane, and the Endothelium.

10. How does the water content of the cornea differ from the rest of the eye?

The cornea is relatively dehydrated, which helps with clarity. If water gets into the cornea, via a disrupted endothelium or from pressure forces with extremely high IOP (acute glaucoma) the cornea turns hazy and white.

11. A pseudophakic patient is found to have excellent far vision, but reading is terrible. What's going on?

As we get older, our natural lenses harden and do not change shape very well … making it hard to accommodate and see near objects. This phenomenon is called presbyopia and is a normal finding in people over 40 years of age. A prosthetic lens is not able to move at all, so all patients (including small children) with implanted plastic lenses need reading glasses to read.

Chapter Three

Glaucoma

The Eyes Have It by Tim Root

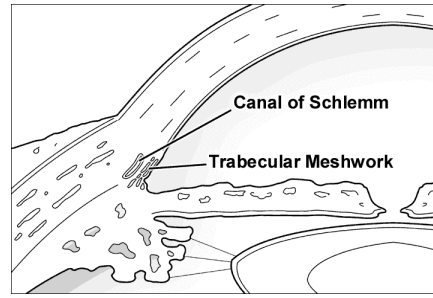
It's just as we feared, Superman …
… you've got optic nerves of steel.

Introduction to Glaucoma

by Tim Root
(last updated 7-3-06)

Glaucoma is a disease where the optic nerve dies. We are not sure *why* or *how* this happens (there are many mechanical, vascular, and biochemical theories) but high intraocular pressure certainly seems to be associated (if not entirely the cause) of optic nerve death. Glaucoma is one of the leading causes of preventable blindness in the U.S. … and patients with *acute* glaucoma can develop irreversible vision loss within a few hours, so it is important that you understand how this disease works and recognize it in your patients.

The Aqueous Pathway



Before continuing we should review the pathway of aqueous humor flow. Aqueous humor is first produced by the ciliary body within the posterior chamber. After filling the posterior chamber, aqueous fluid then moves forward around the lens and flows through the pupil into the anterior chamber. As the anterior chamber fills, the aqueous spreads outwards into the angle formed by the iris and cornea. Within this irido-corneal angle the aqueous exits the eye by filtering through the trabecular meshwork into the Canal of Schlemm, where it enters into the blood circulation. The pressure within the eye is maintained by this steady state of aqueous production and egress.

Open vs. Closed-Angle Glaucoma

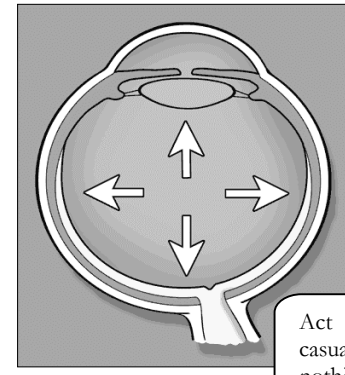
There are two categories of glaucoma and they have very different mechanisms. Open-angle glaucoma is the most common type. It occurs from decreased aqueous drainage caused by a dysfunction or microscopic clogging of the trabecular meshwork. This leads to chronically elevated eye pressure, and over many years, gradual vision loss.

This differs from closed-angle glaucoma, also called “acute glaucoma,” which occurs when the angle between the cornea and iris closes abruptly. With this closure, aqueous fluid can’t access the drainage pathway entirely, causing eye pressure to increase rapidly. This is an ophthalmological emergency and patients can lose all vision in their eye within hours.

Let’s examine each of these types of glaucoma in more detail.

Open-Angle Glaucoma

The majority of glaucoma patients (about 80%) have chronic open angle glaucoma. Most patients are over the age of 40. This condition is much more common in African Americans, and has a strong familial inheritance. Other risk factors include diabetes (debatable), near-sightedness, previous eye injuries, and long-term use of steroids. More recently, thin-corneas have been found to be a major risk factor, though this mechanism is not well understood.



The underlying mechanism for open-angle glaucoma involves degeneration of the trabecular meshwork, usually by unknown causes, that leads to aqueous backup and chronically elevated eye pressure. With prolonged high pressure, the ganglion nerves in the retina (the same nerves that form the optic nerve) atrophy. The exact mechanism for this nerve damage is poorly understood and proposed mechanisms include stretching, vascular compromise, and glutamate transmitter pathways. As the ganglion nerves are progressively destroyed, vision is lost.

Open-angle glaucoma has the reputation of being the "sneaky thief of sight" because the visual loss occurs so slowly that many patients don't realize they have the disease until it is far advanced.

Because the disease is otherwise asymptomatic, detecting open-angle glaucoma requires early pressure screening. Free screening clinics also use different types of automated field testing to detect subtle peripheral vision loss.

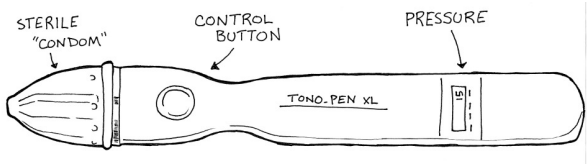
Presentation

Open-angle glaucoma patients usually present with three exam findings: elevated eye pressures, optic disk changes, and repeatable visual field loss patterns.

1. Pressure: The most accurate way to measure eye pressure is with the Goldman applanation tonometer. This is a device mounted on the slit-lamp that measures the force required to flatten a fixed area of the cornea. Normal pressures range from 10 to 20 mm Hg, while glaucoma patients typically measure over 21 mm Hg. Keep in mind that eye pressure can fluctuate throughout the day (typically highest in the morning) so the pressure should be checked with each visit and the time of measurement should be noted. Also, some

glaucomatous eyes have “normal” pressure. A “good pressure” doesn’t rule out glaucoma.

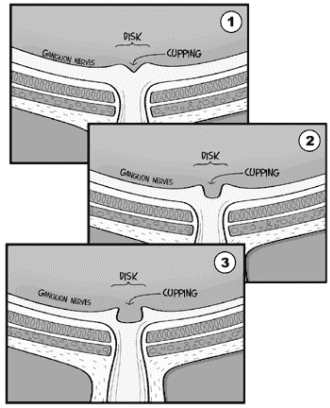
You can also measure pressure with a device called a “Tono-Pen.” This expensive little device is handy for bed-bound patients and down in the emergency room … though it’s inaccurate in the wrong hands.



Corneal Thickness can affect your pressure measurement:

When we measure the pressure in the eye, we are actually measuring how much resistance we get when pressing on the cornea. This is analogous to kicking a car-tire with your foot or pressing your hand against a bicycle tire to estimate how much air pressure is inside. We do the same thing with the Goldman applanation tonometer mounted on the slit-lamp … we measure how much force it takes to flatten a 3mm2 area of corneal surface.

The pressure measurements on the Goldman were calibrated using an average corneal thickness of approximately 540nm. However, some patients have very thin or thick corneas. I like to describe these as “thin bicycle” or “thick truck-tire” corneas. When you press on a thick cornea (a truck-tire cornea) the pressure will seem higher than it really is! This makes sense … if you kick a flat truck-tire, it will still hurt your foot because that rubber is so darn thick. The opposite is true for thin corneas … they are a little squishy no matter how hard you push.



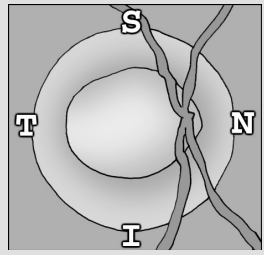
This thickness variability is important in glaucoma clinic so we can calibrate the accuracy of our pressure readings. We always check corneal thickness with an ultrasonic pachymeter on the first visit.

2. Fundus Exam: The optic disk looks striking in these patients. In normal patients, the optic disk has a physiological indentation or “cup” that is less than one-third the disk diameter. With glaucoma, the ganglion nerve layer slowly dies away, and, as fewer ganglion nerves course through the optic disk, the amount of cupping increases. A cup to disk ratio greater than .5 or an asymmetry between the eyes suggests ganglion atrophy caused by glaucoma.

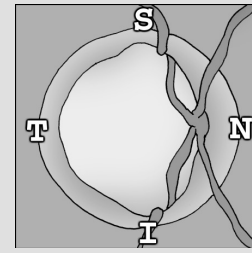
The ISNT Rule

When you look at your patient's optic nerve, you'll notice that the cup doesn't sit directly in the middle of the disk. The cup is slightly off-center – this makes sense as the optic nerve enters the back of the eyeball at an angle.

The space between the inner cup and surrounding disk is called the **neural rim** and is comprised of the actual retinal ganglion nerves. In a normal eye, this rim follows the **ISNT rule** of decreasing thickness … meaning that the **I**nferior rim is thickest while the **T**emporal rim is the skinniest.



normal



abnormal

With glaucoma, you often see vertical thinning and notching of the inferior and superior rims. This deviation from the ISNT rule is another clue that glaucoma might be killing off the nerve. You can also see undermining of the rim (like in the right drawing) where the blood vessels dive out of view under the rim edge.

3. Visual Loss: The vision loss from chronic glaucoma occurs in a characteristic pattern that can be followed on Humphrey visual fields. The central vision is typically spared – in fact, late stage patients may have 20/20 central vision, but be otherwise legally blind because of peripheral blindness.

Fun Fact!

The giant squid has the largest eyeball in the world. This enormous deep-sea predator can weigh up to 2.5 tons and 55 feet in length. Its eye can be a more than a foot in diameter!

Treatment:

Since IOP is the only risk factor we can treat, the treatment of glaucoma focuses on decreasing eye pressure to less than 20 mm Hg. Treatment may be either medical or surgical.

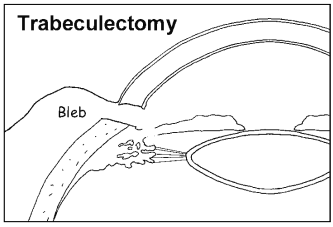
Medical Treatment
Topical beta-blockers are the traditional therapy for these patients and have been around for decades. Beta-blockers work by decreasing aqueous humor production at the ciliary body. Unfortunately, systemic side effects can occur from nasal absorption, making it especially important to ask your patients about history of asthma, COPD, and cardiac problems.

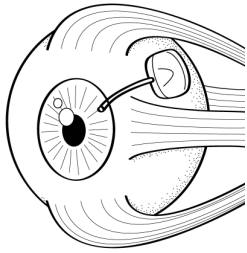
These days, many physicians are using newer drugs like topical CAIs, alpha-agonists, and prostaglandin analogues for first-line therapy, as they have fewer systemic side effects.

Prostaglandin analogues like latanoprost (Xalatan™) are the newest of these glaucoma drugs, and they are becoming very popular. They work by increasing aqueous humor outflow. They do have some side effects, though. They can make eyelashes grow thicker (many patients actually like this), and in a few patients may darken the iris color, turning green and blue eyes brown.

Surgical Treatment for Chronic Glaucoma

If eyedrops aren't working, there are several surgical techniques available to relieve eye pressure. One common surgery is the **trabeculectomy**, where an alternate drainage pathway is surgically created. A small hole is cut through the superior limbus, creating a drainage tract from the anterior chamber to a space under the conjunctiva. This can be very effective in decreasing pressure, but if the patient is a rapid healer the shunt can scar down and close, so anti-metabolites like 5-FU or mitomycin C are often injected at the site. If this surgery doesn't work, a plastic tube can be inserted into the anterior chamber that drains to a plate fixed under the conjunctiva.





Several laser procedures can also help. Argon laser trabeculoplasty (**ALT**) can be used to burn portions of the trabecular meshwork itself. The resulting scarring opens up the meshwork and increases outflow. A laser can also be used to burn away part of the ciliary body to decrease aqueous production.

Acute Glaucoma

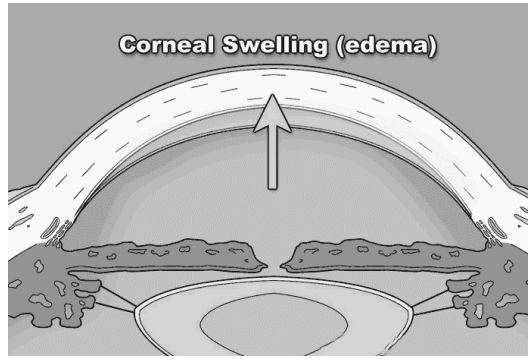
Acute glaucoma is a medical emergency. The most common mechanism is pupillary block. This occurs when aqueous fluid movement is hindered while moving forward through the pupil. This resistance produces a **pressure**

gradient (this is a good buzz word to memorize) across the iris that forces the iris to move anteriorly. When the iris moves forward, the irido-corneal angle closes, blocking the trabecular meshwork. Without an exit pathway, aqueous fluid builds up, eye pressure increases rapidly, and the retina is damaged from stretching and decreased blood supply.

The outflow angle can close for many reasons, and people with naturally shallow anterior chambers, such as hyperopes (far-sighted people) and Asians, are predisposed to developing angle closure. One inciting condition that is typical in acute glaucoma is pupil dilation -- many patients describe onset of their symptoms occurring while in the dark or during stressful situations. When the iris dilates, the iris muscle gets thicker and the irido-corneal angle becomes smaller, making it more likely to spontaneously close. Along those lines, medications that dilate the eye, such as OTC antihistamines and cold medications, also predispose angle closure.

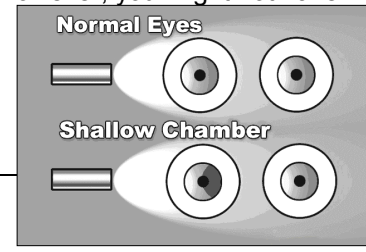
Presentation

These patients will present with an extremely red and painful eye, often complaining of nausea and vomiting. On exam, you'll find their pupil sluggish and mid-dilated. Pressures in the affected eye can be very high, often 60 mm Hg or higher. The eye will feel rock hard, and you can actually palpate the difference between the eyes with your fingers. One classic sign that patients often describe is seeing halos around lights. This occurs because the cornea swells as water is pushed under high pressure through Descemet's membrane into the corneal stroma.



Acute Glaucoma Exam Techniques:

Ophthalmologic examination for acute glaucoma involves measuring the eye pressure, accessing the anterior chamber angle, and a fundus exam. Ideally you want to measure pressure with a slit lamp. However, you might not have access to one in the ER, so you may need to use a Tono-Pen electronic device to measure the pressure.

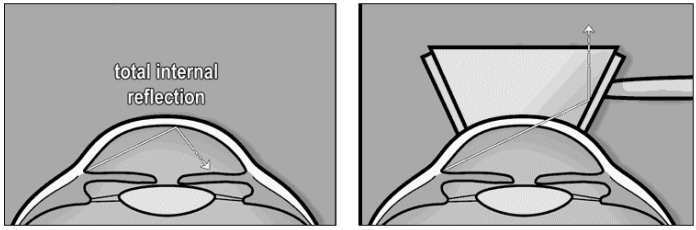


You can determine whether an angle is shallow by shining a simple penlight across

the eyes. If the iris is pushed forward, it will cast a shadow. Additionally, an ophthalmologist can visualize the angle directly through gonioscopy. Here's how it works:

Gonioscopy:

Normally, the inside angle cannot be seen with a slit lamp or magnifying glass because the cornea-air interface creates "total internal reflection." However, an ophthalmologist can use a goniolens, which is a special glass lens with mirrors on its sides to look directly at the angle. When the glass lens is placed directly onto the cornea, the cornea-air interface reflection is broken and light from the angle can escape and be seen through the mirrors.



Fun Fact!

We can see the concept of "total internal reflection" in nature. For example, if you've ever gone snorkeling in the water, you may notice that the water's surface above you looks like a mirror.

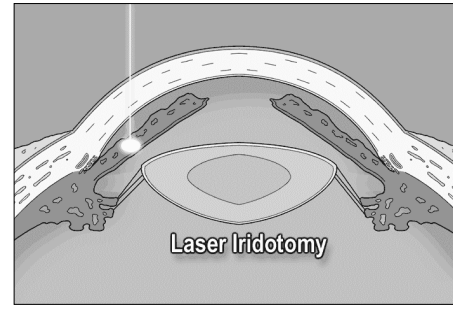
The flying fish uses this phenomenon to escape from predators … when attacked, the flying fish leaps from the water and glides above the surface so the shark can't see them … effectively disappearing.

This is also how fiber optic cables work … light bounces off the walls of the cable.

Acute Glaucoma Treatment

In cases of acute glaucoma, you want to decrease the pressure in the eye as quickly as possible. A "kitchen sink" approach is often used, throwing many treatments on at once. You can decrease aqueous production using a topical beta-blocker like Timolol and a carbonic anhydrase inhibitor like Diamox. Also, osmotic agents such as oral glycerin or IV mannitol (even ethanol, in a bind) can be given that will draw fluid out of the eye and back into the bloodstream. Finally, a miotic, such as pilocarpine, will constrict the pupil and help open up the outflow angle.

Ultimately, these patients need surgical treatment to avoid recurrence of their angle closure. A high intensity laser can burn a hole through the iris and create a communication between the posterior and anterior chambers, relieving the **pressure gradient** (buzzword!!) across the iris, and allowing it to move back into a normal position. This opens up the trabecular meshwork and allows aqueous fluid to flow freely out of the eye. This procedure is typically performed on both eyes because these patients are predisposed to having attacks in the other eye as well.



Other types of glaucoma

1. Neovascular Glaucoma:
This can occur in diabetic patients or those with an old retinal vascular occlusion. VEGF production from areas of ischemic retina float forward through the pupil and promote neovascularization of the iris. In the early stages, a fibrous membrane forms on the iris-cornea angle that blocks outflow and forms an open-angle glaucoma. At later stages of neovascularization, the new vessels actually pull the iris forward and cause a closed angle glaucoma that is essentially irreversible. You treat this by lasering the peripheral retina to decrease the angiogenic VEGF production and decrease the rate of neovascularization. Most of these patients end up needing a surgical intervention.

2. Pigment Dispersion Syndrome (PDS):
Occurs when the pigmented back-surface of the iris rubs against the radial zonules supporting the lens. Little flecks of pigment are shed into the aqueous and end up clogging the trabecular meshwork drain. Victims are usually young white males and can suffer from attacks of high pressure after exercise. You can see pigment in the TM via gonioscopy, and you can also see trans-illumination defects in the thin iris itself.

Some of the pigment will stick to the inner-corneal surface, and because of convection currents in the aqueous, form a vertical line of pigment called a Krukenberg spindle.

3. Pseudoexfoliation Syndrome (PXF)
In this condition, systemic basement membrane-like material is deposited throughout the body. This material adheres to the anterior lens capsule, creating a rough surface. As the overlying iris dilates and contracts with daily activity, pigment is rubbed off and clogs the

trabecular drain. These patients also suffer from zonular instability, making cataract operations difficult.

Summary

That is glaucoma in a nutshell. Chronic, open-angle glaucoma is very common (in this country) and leads to gradual visual loss, while acute closed-angle glaucoma is infrequent but an emergency that needs urgent treatment to avoid blindness.

1. What is glaucoma? What actually causes damage to the neurons and optic nerve with glaucoma?

Nobody is sure exactly "what glaucoma is" … but at its root, glaucoma is gradual death of the optic nerve. If an attending asks you, say "death of the optic nerve." If you say "high pressure" you'll be laughed at (glaucoma specialists are odd ducks). The optic nerve damage could come from pressure, stretching, sheer forces, or some kind of hormone regulator … we're not sure.

2. What's the difference between open-angle and closed-angle glaucoma? Also, how about chronic versus acute glaucoma?

Open angle is a common, chronic condition where aqueous drainage is impaired. Closed-angle glaucoma is caused by acute closure of the irido-corneal angle, and is an ophthalmologic emergency that can quickly lead to blindness.

3. What are the risk factors for developing primary open-angle glaucoma?

This will definitely be on your test. Since I didn't list them well in the chapter, here they are. These are the risk factors as determined by the OHTS trial, one of the biggest glaucoma trials.

High Intraocular Pressure (obviously)
Age
Family History
Race (African American and Hispanics)
Suspicious Optic Nerve Appearance (large vertical cupping)
Thin Corneas (** remember this one!)

There are other possible risk factors, such as high myopia and *possibly* diabetes, but I'd focus on those above.

4. What do we measure to monitor and access progression in glaucoma patients?
We generally check three things: pressure, disk changes by 3D photograph, and visual fields.

5. What's a normal eye pressure? Does a patient with pressure of 14 have glaucoma?
About 10 to 20. People can still have glaucoma with normal pressure. Also, pressure fluctuates throughout the day and we typically write down the time in the notes.

6. A glaucoma suspect is found on first visit to have a pressure of 18. Her corneal thickness, however, measures only 450nm. Do you think her actual eye pressure is HIGHER or LOWER than 18?

Definitely higher. This patient has thin "bicycle-tire corneas" that "feel soft" when measured by the Goldman tonometer. This woman's pressure is probably well over 20, increasing her risk for glaucomatous damage progression.

7. What does corneal thickness have to do with glaucoma (as far as risk for developing glaucoma)?
The OHTS trial showed that people with thin corneas are at higher risk for glaucoma, independent of other risk factors. We're not sure why, but it's believed that people with thin corneas are anatomically predisposed to optic nerve damage. With all new glaucoma screens, we measure corneal thickness with a small ultrasound probe (this is called pachymetry).

8. What kind of vision loss does glaucoma cause?
Typically loss of eyesight occurs in the periphery first where the loss is less noticeable. Many patients don't notice visual symptoms until the disease is far progressed. Scotomas (visual field loss areas) in glaucoma tend to follow certain patterns. Generally, the central vision is spared until very late stages of glaucoma.

9. What is the flow-pathway for aqueous fluid? Where is it made, and where does it leave the eye?
Aqueous is produced by the ciliary body, then it flows forward through the pupil into the anterior chamber. Finally, it drains through the trabecular meshwork and into the canal of Schlemm back in the venous system.

10. How do you visualize the aqueous drainage pathway in the eye?
Gonioscopy allows direct visualization of the trabecular meshwork.

11. Why can't you see the trabecular meshwork with the slit-lamp microscope?
Because the trabecular drain is behind the limbus (around the corner) and we can't see there directly because of "total internal reflection" … memorize this phrase and use it in clinic and your attending will be impressed!

12. What mechanisms do the glaucoma drops use to decrease pressure?
Drops either decrease the amount of aqueous produced or increase the aqueous outflow from the eye.

13. What retina findings do you see with glaucoma?
You see cupping of the optic disk. You can sometimes see hemorrhages at the disk and undermining of the vessels as they leave the disk.

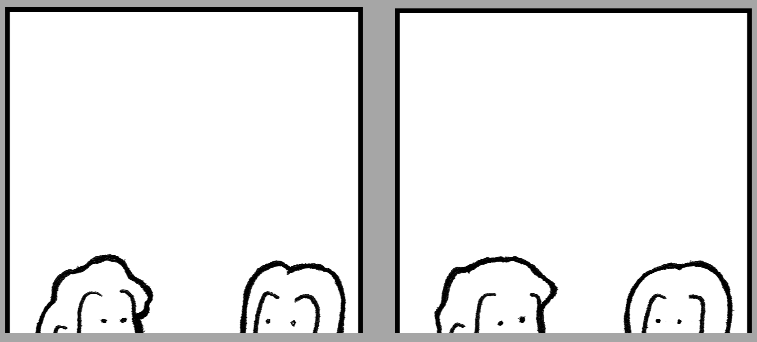
14. How can diabetes cause acute glaucoma?
Retinal ischemia can cause neovascularization of the iris … forming a membrane that covers TM or closes the drainage angle. This leads to a serious glaucoma that is hard to manage.

15. You have a patient who appears to have a shallow anterior chambers and subsequent occludable angles. Would you use pilocarpine?

In most cases, yes. Pilocarpine will constrict the pupils -- by flattening the iris you potentially open up the drainage angle. Pilocarpine will also decrease pressure in the eye by a couple of mechanisms. You probably wouldn't use it long term in patients with occludable angles though, as pilo has a lot of side effects. Ultimately, anyone with occludable angles needs a peripheral iridotomy to equalize the pressure between anterior and posterior chambers. "Equalize pressure" … that's another buzz phrase you should memorize.

Chapter Four

Diabetes

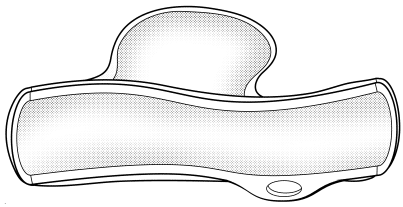


Diabetic Retinopathy

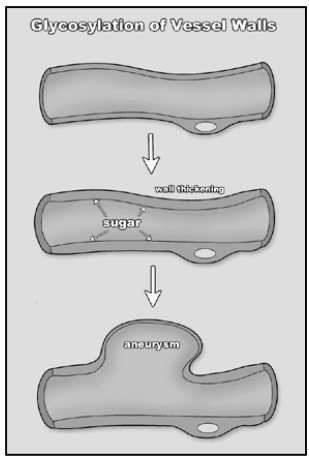
by Tim Root
(last updated 7-3-06)

Diabetes is a common disease and many affected patients have vision problems. In fact, diabetics are twenty times more likely to go blind than the general population. **Diabetic retinopathy** is the term used to describe the retinal damage causing this visual loss. Diabetics have a high prevalence of retinopathy, and one out of every five patients with newly diagnosed diabetes will also show signs of retinopathy on exam.

Mechanism of Vessel Breakdown:



How are the eyes affected? Basically, diabetes is a disease of blood vessels. With large amounts of glucose coursing through the circulatory system, a **glycosylation reaction** occurs between the sugar and the proteins that make up the vessel walls. Over time, this glycosylation promotes denaturing of collagen protein within the walls, creating capillary thickening and eventually, wall breakdown.



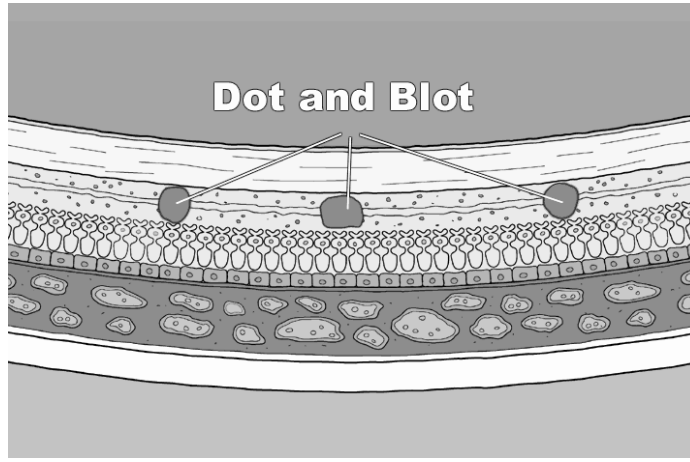
While this process occurs throughout the entire body, the microvasculature of certain organs, such as the kidneys and eyes, are more susceptible to damage. Along those lines, a good predictor of microvasculature damage in the diabetic eye is prior evidence of renal microvasculature disease as measured by proteinurea, elevated BUN and creatinine.

Because vessel damage accumulates over time, the most accurate predictor of retinopathy is duration of diabetes. After 10 years, more than half of patients will show signs of retinopathy, and after 15 years this number increases to nearly 90%. The relative control of glucose during this time is also important, and studies have shown that patients who maintain lower hemoglobin A1C levels have delayed onset and slower progression of disease. Additional risk factors include smoking, hypertension and pregnancy.

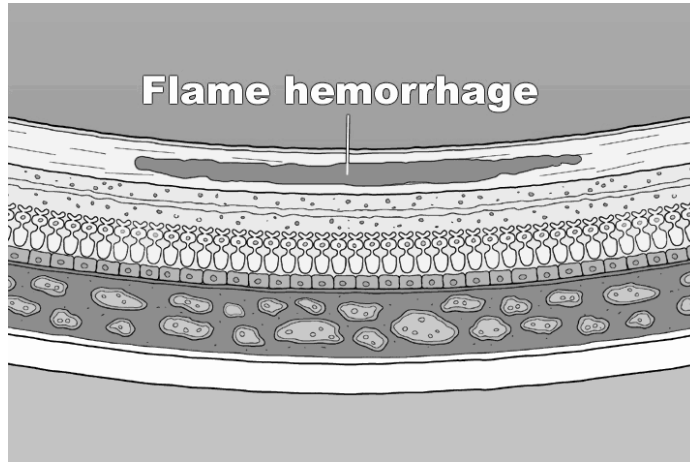
Two Types of Retinopathy:

It is useful to divide patients into two categories of retinopathy.

A. Nonproliferative diabetic retinopathy (NPDR): Most patients (95%) have NPDR. This is the earliest stage of retinopathy and it progresses slowly. Because so many diabetic patients have NPDR, this stage is commonly described as "background retinopathy." The earliest signs of retinal damage arise from capillary wall breakdown, seen on the fundus exam as vessel microaneurysms (or "outpouchings"). Injured capillaries can leak fluid into the retina and the aneurysms themselves can burst, forming **"dot-and-blot hemorrhages."**



Dot-and-blot hemorrhages look small and round because they occur in the deep, longitudinally-oriented cell layers of the retina. This contrasts with the "flame hemorrhages" of hypertension that occur within the superficial ganglion nerve layer, and thus spread horizontally.

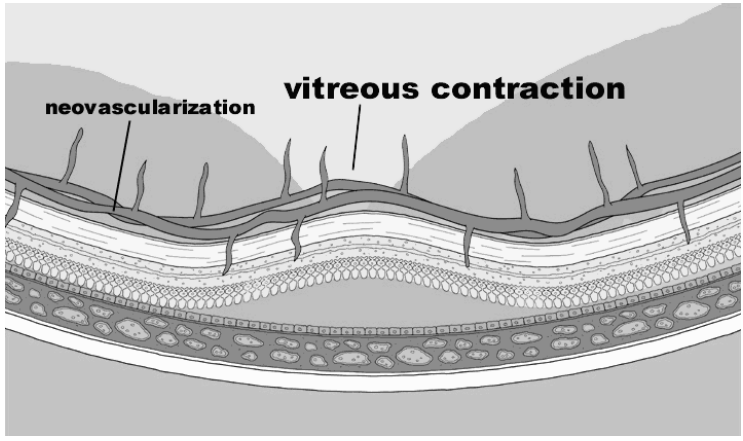


With worsening retinopathy and vessel damage, the retina begins to show early signs of ischemia. **Cotton-wool spots**, also called "soft exudates," are gray spots with soft edges that indicate ischemia/infarction of the retinal nerve fibers. As vessel damage progresses, you can also see beading of the retinal veins.

B. Proliferative Retinopathy – With ongoing injury to the retinal vasculature, there eventually comes a time when the vessels occlude entirely, shutting down all blood supply to areas of the retina. In response, the ischemic retina sends out chemicals that stimulate growth of new vessels. This new vessel growth is called **neovascularization**, and is the defining characteristic of proliferative retinopathy. Far fewer patients have proliferative retinopathy, which is fortunate as this stage can advance rapidly, and half these patients will go blind within five years if left untreated. The mechanism and complications of neovascularization merit study, so let's take a closer look.

The Mechanism of Neovascularization:

With complete vessel occlusion, parts of the retina become starved for nourishment. The ischemic retina responds by releasing angiogenic molecules like VEGF to promote new vessel growth. These new vessels serve to bypass the clogged arteries in order to resupply the starved retina.

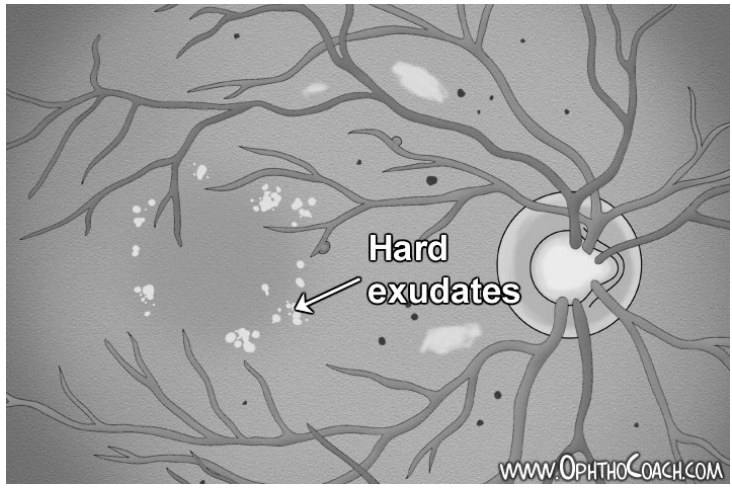


A collateral blood supply seems like a great idea, but unfortunately there is a problem. The newly formed vessels are abnormal in both appearance and function. The new vessels are friable and prone to leaking. They also grow in the wrong place, spreading and growing along the surface of the retina. They can even grow *off* the retina, sprouting up into the vitreous fluid. The vitreous is mostly water, but it also contains a lattice framework of proteins that the new vessels can adhere to. With vitreous movement or contraction, these new connections pull on the retina and the traction can create a detachment. Since the vessels are also weak, any vitreous traction can break them and

create sudden hemorrhaging with subsequent vision loss as the eye fills with blood. Finally, the new vessels can regress and scar down, creating massive traction on the retina underneath.

Neovascularization isn't just limited to the retina, but can also occur on the iris itself. **NVI (neovascularization of the iris)** is an ominous sign, as the new vessels can cover the trabecular meshwork and create a sudden neovascular glaucoma.

Macular Edema
Despite the neovascularization phenomenon and its potential for detachments and hemorrhaging, the most common cause of blindness in diabetic patients is from macular edema. This occurs when diffuse capillary and microaneurysm leakage at the macula causes the macular retina to swell with fluid.



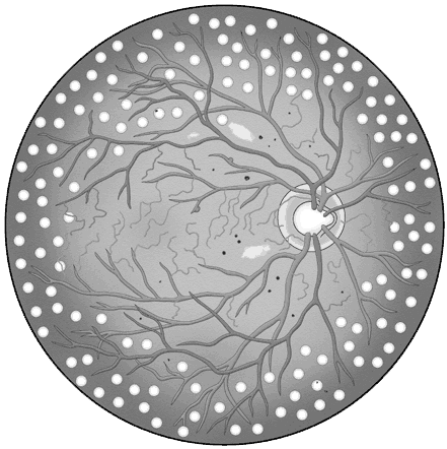
Macular edema occurs in about 10% of patients with diabetic retinopathy and is more common with severe retinopathy. On exam the macula looks cloudy, with microaneurysm, and you can see past evidence of edema in the form of yellow-colored **"hard exudates"**. These exudates are fatty lipids that get left behind when past macular swelling subsides, similar to a dirt ring in a bathtub.

Treatment of DR (diabetic retionopathy):

Preventative medicine with tighter control of glucose is the ideal treatment, but for worsening symptoms, surgical treatment is necessary. The two main surgeries are laser treatment and vitrectomy.

Laser Treatment
In cases of macular edema, an argon laser can be used to seal off leaking vessels and microaneurysm in the retina by burning them. If the leakage or microaneurysm is small and well-defined, it can be

selectively sealed off. With larger areas of leaking capillaries, such as diffuse macular edema, the laser can lay down a “grid photocoagulation” pattern over the entire area.



With advanced retinopathy and neovascularization, a different approach is taken. Instead of individually targeting vessels, **PRP (pan-retinal photocoagulation)** is performed. With PRP, the ophthalmologist burns thousands of spots around the peripheral retina. This destroys the ischemic retina, decreasing the angiogenic stimulus, and commonly leads to regression and even the complete disappearance of the new vessels. This treatment may seem drastic, but it has proven to be effective. Naturally, there are side effects, with peripheral vision loss and decreased night vision (from the rod photoreceptor loss), but this is acceptable if the central vision is saved.

Vitrectomy

A vitrectomy may also need to be performed and is often done in conjunction with other surgeries. This surgery involves removing the vitreous humor from the eye and replacing it with saline. This removes hemorrhaged blood, inflammatory cells, and other debris that may obscure the visual axis. While removing the vitreous, the surgeon also removes any fine strands of vitreous attached to the retina in order to relieve traction that might have caused a detachment.

Conclusion:

As you can see, diabetic retinopathy is a big problem … and a large percentage of our retina patients have diabetes. Retinal vessel damage leads to edema, and vessel occlusion stimulates neovascularization that can quickly lead to trouble. Fortunately, better glucose control and surgical treatments have significantly decreased the incidence of visual loss in these patients.

Pimp Questions

1. What is diabetic retinopathy, and by what mechanism does it occur?
This is change in the retina, secondary to leaky blood vessels.

2. What are the retinal signs of diabetic retinopathy. How do they compare, to say, hypertension retinopathy.
You typically get a lot of dot-blot hemorrhages. HTN usually has more flame hemorrhages. You can also see cotton wool spots and hard exudates.

3. How are angiogenic molecules involved with diabetic retinas?
VEGF production by areas of ischemic retina leads to neovascularization … which is bad, as they can cause traction, more leakage, detachments, etc.

4. How do we categorize diabetic retinopathy?
As either NPDR (nonproliferative diabetic retinopathy) or PDR (proliferative retinopathy)

5. What are some mechanisms in diabetic retinopathy that might lead to decreased vision? This will probably be on your test.
There are several mechanisms for potential vision loss in these patients, including:

Macular edema (probably the #1cause of decreased vision)
Vitreous hemorrhage
Retinal detachments
(dot-blots don't really cause decrease in vision)

6. How do we treat advanced diabetic retinopathy?
PRP (pan retinal photocoagulation). By killing off the peripheral ischemic retina, we decrease VEGF production and decrease neovascularization.

7. A 35 year old man with bad Type 1 diabetes presents with a pressure of 65. His anterior chamber is deep but you find neovascularization everywhere … in the retina and on the iris. What do you think is causing the pressure rise, and how do you treat it?

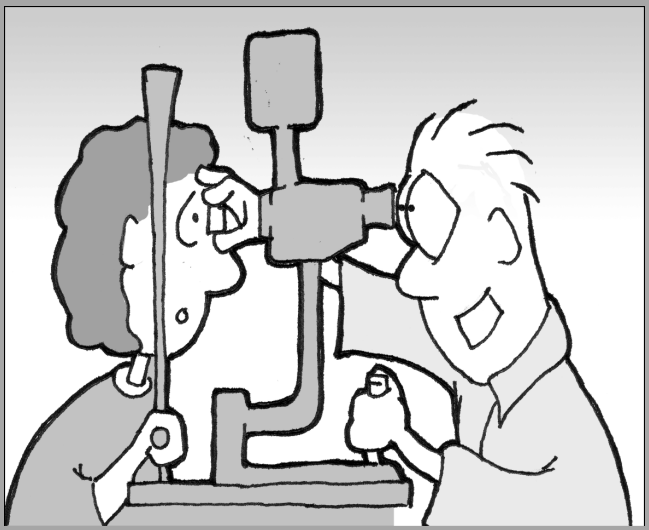
The pressure is up because of neovascularization on the angle … blood vessels have clogged up the trabecular drain. You treat neovascularization by PRP lasering the peripheral retina to decrease VEGF production. NVA is

hard to manage, though, and this patient will probably require a surgical drainage procedure.

Chapter Five

Retina

The Eyes Have It by Tim Root



Well, they say the eye is the "window to the soul" …
… and your soul appears to be swollen, bloody, and detached.

Retina

by Tim Root
(last updated 7-3-06)

The retina can be intimidating … there is a bunch of pathology back in the retina, and the anatomy is not easy to visualize (especially as a medical student or non-ophtho resident rotating through our clinic!). There are many things I could cover in this chapter, but I've decided to keep things simple and only discuss a few topics like retinal detachments. Other disease processes that involve the retina (such as CMV retinitis) I've covered in other chapters.

Famous Quotes

Once while we were making love, a curious optical illusion occurred, and it almost looked as though she were moving.

Woody Allen

Retinal Detachment

A retinal detachment is an abnormal separation between the sensory retina and the underlying RPE and choroid plexus. Without the supportive nourishment from the choroidal blood vessels, the retina becomes ischemic and begins to necrose. The macular retina is especially susceptible to this ischemia. The prognosis for patients with retinal detachments depends upon the quickness to treatment; patients with detachments that involve the macula have much worse outcomes.

Risk Factors and Epidemiology:

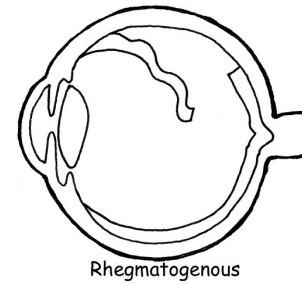
Up to six percent of the general population have retinal breaks of some kind, though most of these are benign atrophic holes. The actual incidence of retinal detachment is only 1 in every 10,000 people. Relative risk is equal between men and women, with higher rates in those of Jewish descent and decreased risk in black populations.

When looking at patients who already have retinal detachments, you begin to see some interesting trends. To begin with, nearly half of these patients are myopic (near-sighted). Myopic eyes are physically larger and longer than normal eyes and have thinner retinas at the periphery. This thin retina is more likely to break down, forming small holes and tears that may progress to a detachment.

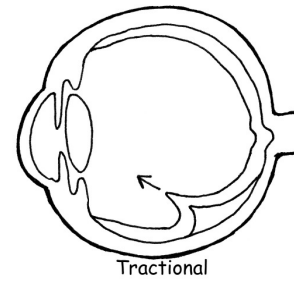
Up to 35 percent of patients with retinal detachments develop them after another eye surgery – typically a cataract extraction. Finally, traumatic sports such as boxing, football, and bungee-jumping predispose younger people to forming detachments.

The Three Types of Detachment
Retinal detachments generally occur by three different mechanisms.

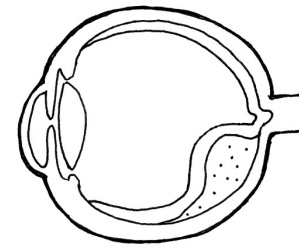
1. The most common detachment is the **rhegmatogenous retinal detachment**. This is an actual tear in the retina, with a full-thickness break through the retinal sensory layers. These tears can occur from trauma or surgery, or extend from preexisting retinal holes. Fluid from the vitreous chamber flows through the tear, and collects in the sub-retinal space. Eventually, the retina tears away, peeling off the RPE and the choroid underneath. Without treatment, the detachment will spread and involve the entire retina.


Rhegmatogenous

2. The second category is detachment from **traction** of the retina. This is when the retina is pulled off its anchor from above. It can occur from vitreous contractions, or from diseases such as diabetic retinopathy where neovascular membranes on the retinal surface contract and pull on the retina with great force.


Tractional

3. A less common mechanism for detachment is from **hemorrhagic** or **exudative retinal detachment**. This occurs when blood or fluid builds up under the retina, slowly pushing the retina upwards. This occurs with dysfunction of the RPE or choroid plexus, caused by ocular tumors, inflammatory diseases, or congenital abnormalities that create a breakdown of the blood-retina barrier.


Exudative

PVD (posterior vitreous detachment)
One common cause of a retinal tear is secondary to a posterior vitreous separation. As we age, the vitreous liquefies and contracts in on itself … if this occurs suddenly, the posterior vitreous face can suddenly pull off the retina. Usually, this isn't a problem, but if the vitreous is abnormally adherent

to the retina the separation may rip a small hole in the retina that progresses into a detachment.

PVDs are very common in people over 65 and are a major source of annoying floaters" in this population.

Symptoms:

With detachment, patients often report seeing flashes of light and floaters.

Flashing lights, or **photopsia**, is often seen when the detachment first occurs. Photoreceptors are normally triggered by light, but severe mechanical disturbance can stimulate them as well, and give the sensation of light like a camera flash.

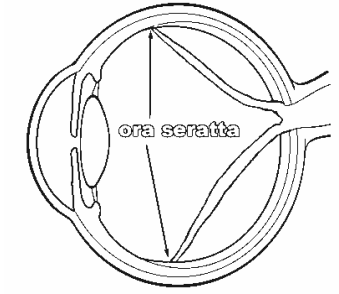
Floaters look like dark specks that obscure vision, and patients say they look like a swarm of flies. They are created by objects (blood cells or pigment) floating in the vitreous chamber that cast shadows on the retina. While the presence of a few floaters is normal, the sudden appearance of hundred of floaters indicates a bloody hemorrhage into the vitreous.

The combination of flashing lights and floaters should be considered a retinal detachment until proven otherwise. Another symptom that is sometimes described is seeing a **"dark curtain"** that obscures peripheral vision, as most detachments occur in the peripheral retina.

Findings:

The definitive way to diagnose a retinal detachment is to actually see it with the indirect ophthalmoscope. If the tear is large enough, it will be obvious as it undulates with eye movement. Suspended pigment particles may be seen floating in the anterior vitreous (**Shafer's sign**), described as "tobacco dust," and is pathognomonic for a retinal tear.

An ultrasound of the eye may be helpful, especially when the tear is not obvious or when the retina can't be seen because of hemorrhaging or cataracts. An ultrasound can also pick up other pathology such as tumors.



This illustration shows an ultrasound of a patient with a complete retinal detachment. The retina looks like a letter V in this picture, because it is still attached at two places – the optic disk and in the peripheral ora seratta. Choroidal effusions also give this

appearance, but I won't talk about them because it would just be confusing at this point.

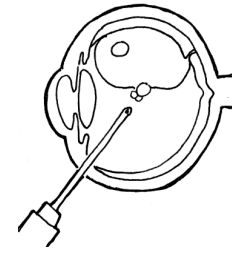
Treatment Options:

The treatment for retinal detachment varies. Exudative detachments are treated much differently, with treatment directed toward the underlying cause of the exudate, such as a tumor or inflammation. However, the primary treatment for the majority of retinal tears and traction detachments is surgical. How fast a patient needs surgery depends upon whether the central macula has detached or not. If the macula has detached, then the vision is pretty much toast anyway, so you can wait a few days before going to surgery.

If the retina has a tear or hole that hasn't yet detached, the tear can be "pegged down" by welding down the surrounding retina with a **laser**. The retina can also be scarred down by freezing it into place with a **cryoprobe** applied from the outside of the eye.

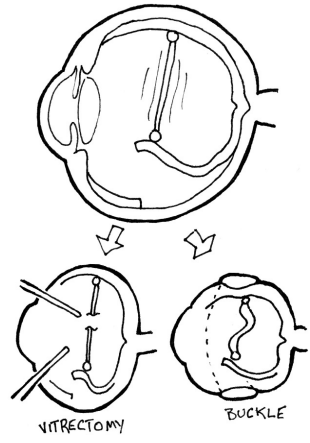
Scleral buckling is the traditional surgical procedure, and involves encircling the eye with a silicone band that squeezes the eye like a belt. The buckle indents the eye and pushes the RPE into contact with the retina, allowing it to heal into place. Because of the orbital anatomy, scleral buckles are most useful in anterior breaks because you can't really buckle the back of the eye.

Over the past two decades, **pneumatic retinopexy** has become quite popular. In this procedure, after repairing the retinal tear, the surgeon injects a bubble of gas or silicon oil into the globe, which acts to push (or tamponade) the retina into position until it heals. There are many different types of gas that we use, but they all eventually absorb back into the body, and patients have to keep their head down for several weeks to keep the bubble in place. An oil bubble doesn't require this head positioning, but does require a return to the OR to remove the oil.



If the detachment if severe and complicated, a **vitrectomy** may need to be done. The vitreous fluid is removed, and the retina is manually floated back into position. With access to the inner globe, scar tissue and any other causes of traction, such as the neovascular membranes, can be removed.

Rubber Band Theory:
When treating a retinal detachment, a good way to think about traction is the "rubber band" theory. Thus, there is some tension inside the eye that is keeping the retina from laying flat. There are two ways to relieve this tension: you can perform a vitrectomy and "cut" the band, or you can perform an encircling buckle procedure to shorten the band



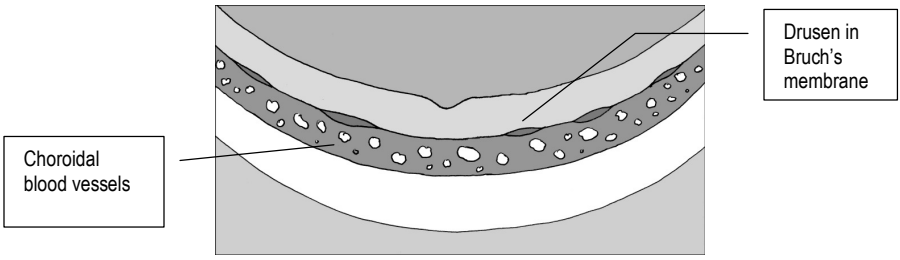
RD Summary:
Retinal detachments used to be universally blinding, but with modern surgical techniques, sight can now be saved. If you suspect a retinal detachment in your patients, send them to an ophthalmologist right away, as their prognosis depends upon the speed in seeking treatment.

ARMD

ARMD stands for Age Related Macular Degeneration and is a common retinal finding in older patients. ARMD is actually the leading cause of blindness in the elderly … at least in developed countries like the US.

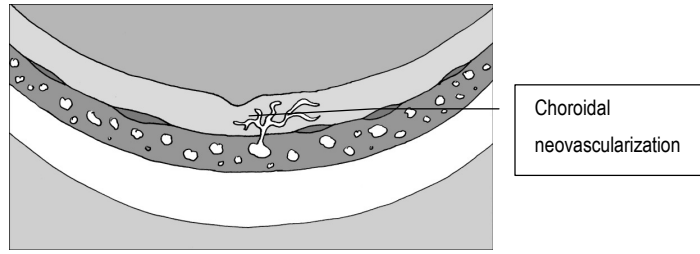
These patients develop extracellular breakdown deposits called "drusen" that form deep in Bruch's membrane. Bruch's membrane is the thin layer that separates the RPE/Retina from the underlying choroidal blood supply. This blockage keeps nutrition from percolating up from the choroid to the retina, and conversely, blocks photoreceptor waste products from draining down into the choroid vessels.

On exam you see localized retinal atrophy and pigmentary changes in the macula that correlate with poor central vision. The visual loss occurs slowly, however, and takes many years to progress.



Neovascular "wet" ARMD

If a break occurs in Bruchs membrane, vessels can grow up out of the deep choroidal circulation directly up into the retina! This is dangerous, as this neovascularization can bleed, create edema from leaking, and rapidly destroy vision.



Treating this macular neovascularization is tricky - we would love to burn it away with a laser … but those bad blood vessels are right at the fovea … and you don't want to burn away central vision! Instead, we can use a few other techniques with variable success:

> **PDT (photodynamic therapy):** We inject a special chemical into the blood that reacts to specific wavelengths of light. Once the chemical floats within the retinal blood vessels, we then focus light of that desired wavelength directly at the fovea to coagulate the blood vessels without destroying the retina around it. Sounds good in theory, but it doesn't work that great.
>
> **Macugen:** We can also inject anti-VEGF drugs like Macugen into the eye to stop angiogenisis (this is what we do in our clinic). Some doctors use off-label Avastatin (the anti-VEGF drug used in colon cancer), as it is much cheaper on a per-dose basis. Newer anti-VEGF drugs are being developed that may work even better.

Monitoring progression

Early, dry ARMD is very common and requires no treatment (other than possibly antioxidant vitamins), but we want to monitor these patients for progression to wet-ARMD. Patients can monitor themselves with an Amsler grid … a sheet of straight lines they can look at weekly to look for metamorphopsia that might indicate macular edema.

Risk Factors?

So who gets ARMD? You see it often in elderly Caucasian patients, who often have a positive family history for the condition. It's almost always bilateral. The disease is also highly associated with smoking.

1. Describe the three kinds of retinal detachment?

They are rhegmatogenous detachments, tractional detachments, and exudative detachments.

2. What are the symptoms of a retinal tear or detachment?
Flashes and floaters are the classic signs. If there is a large detachment your patient may also notice an area of "dark curtain" or decreased peripheral vision.

3. What is a PVD?
This is a posterior vitreous detachment … with aging the vitreous jelly liquefies and contracts. A sudden contraction can cause new floaters. This event is usually harmless, but you should search carefully for retinal tears.

4. An elderly patient presents with a brief episode of flashing, and now with a single floater that moves with eye movement. A thorough retina exam reveals no detachment or tear, but you observe a small vitreous opacity over the optic disk. What has happened?
This sounds like a PVD. The floater is a Weis ring, a piece of optic disk material that has pulled off with the detachment. PVDs are common and harmless, though patients should have a thorough exam for retinal tears and given a description of RD symptoms.

5. A patient presents late at night with a large bullous retinal detachment. The central fovea is also detached. How soon do you need to go to surgery … do you need to wake up your retina attending?

If the macula is off, then you can take your time going to surgery and schedule it a few days later, as the visual prognosis is poor. However, if the macula is still ON, you want to go to surgery right away to make sure it STAYS on.

6. What kind of surgeries can we perform to relieve retinal detachments?

You can do a vitrectomy to clean out the inside of the eye and relieve retinal traction. While in there you can also reappose the retina. You can also perform a scleral buckle or a pneumatic retinopexy.

7. What is Schafer's Sign?
Pigment particles floating in the anterior vitreous chamber, right behind the lens, seen on slit-lamp. This sign increases your suspicion for a tear or detachment.

8. What kind of travel restrictions would you tell a patient who has a pneumatic retinopexy?

Well … you don't want these patients to fly. A decrease in ambient pressure causes gases in the eye to expand. If this happens in the eye it could explode! Your patients should also avoid SCUBA diving and base-jumping.

9. What's the difference between dry and wet age-related macular degeneration?

Dry ARMD is when you have drusen and macular RPE atrophy … "wet" ARMD implies choroidal neovascularization through Bruchs membrane.

Chapter Six

Eye Infections

Eye Infections

by Tim Root
(last updated 7-4-06)

The eye is well protected from infection by the conjunctiva and the corneal epithelium. In addition, the tear film contains antimicrobials and the tear flow itself tends to wash away pathogens. The eye also harbors a host of non-pathogenic bacteria that competitively prohibit new bacteria growth. However, these eye-defenses can be breached by trauma, improper tearing, or contact lens wear and lead to an infection. An eye infection not only threatens vision, but the orbit can act as an entry portal to the rest of the body and infections can progress to systemic infection or meningitis.

Infections are a pain in the butt in the walk-in clinic … we see a lot of conjunctivitis, blepharitis, and corneal ulcers. Here's a review of the common, less common, and potentially devastating infections you should know about.

Pink Eye … the three types of conjunctivitis:

The conjunctiva is the semi-transparent covering over the eye that protects the eye from foreign bodies, infections, and irritants. However, the conjunctiva itself is susceptible to irritation and infection from viruses and bacteria. Conjunctivitis, or "pink eye," is the term used to describe inflammation of the conjunctiva and commonly occurs from three different sources: virus, bacteria, or allergy.

1. Viral conjunctivitis is the most common type, making up half of all cases of conjunctivitis. It is usually caused by an adenovirus, often following an upper respiratory infection or cold. Viral conjunctivitis is quite contagious and other family members may also complain of having "red eye." Infected patients typically present with eye redness and watery *tearing*, but little mucous discharge. Often, only one eye is infected, but the infection may hop to the other side before the end. Two specific signs on exam are enlarged *follicular* bumps on the inside of the eyelids (these look like blisters) and swelling of the preauricular *node* located in front of the ear. Most of these infections clear up on their own within a few days. Like the common cold, treatment is geared toward relieving symptoms. Viral conjunctivitis is so contagious that I also recommend good hygiene and no towel/makeup sharing. A lot of people at our hospital present with pink-eye, and this diagnosis is often a three-day vacation from work for them as we don't want to spread the infection to our patients.

2. Bacterial conjunctivitis presents with a *mucupurulent* (pus) discharge. This creamy discharge may cause your patient to complain of sticky eyelashes, with patients finding their eyes matted shut when waking in the morning. Bacterial conjunctivitis often

develops a papillary conjunctival reaction, and, unlike viral infections, typically does NOT have preauricular node enlargement because the infection drains out the nasolacrimal system. The most common culprits are staph or strep, although with children you should also consider the Hemophilus influenza bacteria. In addition, with sexually active adults you should consider chlamydial and gonococcal infections (especially with severe findings or sudden onset of infection). We treat most conjunctivitis with erythromycin ointment.

3. Allergic Conjunctivitis: Finally, patients with allergic conjunctivitis present with red, watery eyes. The hallmark symptoms of allergy is itching and swelling. Patients often have a history of seasonal allergies and will usually present with other allergic symptoms such as a stuffy nose and cough. Treatment for allergic conjunctivitis involves avoidance of the offending allergens. These patients may need antihistamines, mast-cell stabilizers, and possibly steroids

Pink Eye … what's causing it?
The cause of a conjunctivitis is not always apparent. Typically, you treat with cool compresses, Tylenol, and vigorous hand-washing. If you suspect bacteria, you treat with an antibiotic like erythromycin. Pathognomonic symptoms include:

1. Viral: watering, follicles, swollen lymph nodes
2. Bacterial: creamy dischargej, unilateral
3. Allergy: Bilateral itching and swelling

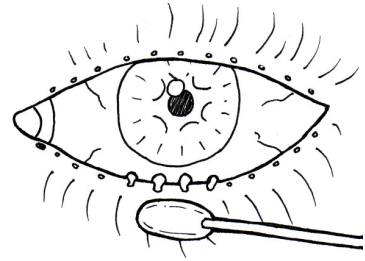
Blepharitis:

The term blepharitis describes an inflammation or infection of the eyelid margin. This is probably the most common finding and diagnosis in our eye clinic, with patients complaining of chronic eye stinging, tearing, and "gritty" sensation of the eyes. Blepharitis has been classified many ways (example: seborrheic blepharitis, staphylococcal blepharitis, etc.). I prefer to distinguish it as either (or a combination of):

A. Anterior blepharitis:
With these patients you'll find debris, "scurf", or collarets at the bases of the eyelashes. If severe, you can see small ulcerations and eyelash loss in some areas.

B. Posterior blepharitis:
This is when the meibomian gland orifices are clogged up. When examining the eyelids, I always push on the lid edges with a Q-Tip. Pus will often come out of these pores. I usually note this in the chart as MGD (meibomian gland dysfunction)

The primary treatment for blepharitis involves good lid hygiene. Most cases can be relieved in a few weeks by having your patient wash their eyelashes daily with baby shampoo and a washcloth. Warm compresses will also help as they open up the orifices of the meibomian glands. Tougher cases of blepharitis may require topical antibiotics. You can also use oral doxycycline – which works not by its antibiotic effect, but by changing the fatty acid oil composition of the meibomian glands.

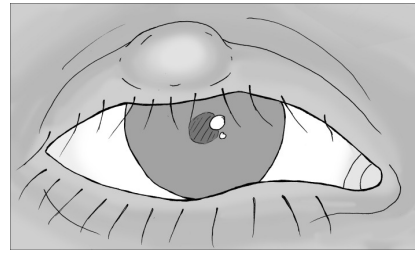


Blepharitis is a chronic condition and almost half of my patients in the on-call clinic suffer some degree of blepharitis. Compresses and lid scrub regimens may need to be continued indefinitely.

Fun Fact!

In addition to long lashes, camels have an extra eyelid to protect their corneas from blowing desert sands. This eyelid is so thin that the camel can close the lid and still see through it - helpful when traveling through sandstorms.

Chalazion:



Chalazions are granulomatous inflammations of the **meibomion gland.** These glands produce the lipid component of the tear film and are deeply located within the supporting tarsal plate of the lid. Chalazions occur when meibomian gland pores become clogged (such as in blepharitis) -- lipid backs up into the gland, and a *noninfectious* inflammatory granuloma reaction occurs.

On exam, the patient will have a firm, nodular swollen area on their lid … and when you evert the lid, you'll often see the chalazion bump more clearly. They are non-tender and are not painful.

Early treatment involves warm compresses, massage, and lid scrubs in an attempt to reopen the meibomian pore and get the material to flow out. If this doesn't work, we flip the lid and incise/drain the chalazion, or sometimes inject steroids. Some people are more prone to developing them, and they tend to reoccur.

Chlamydial Conjunctivitis:

Chlamydia causes two different kinds of conjunctivitis: inclusion conjunctivitis and trachoma. Both of these infections are caused by different serotypes of chlamydia. We don't see these infections often (I've not seen one this entire year), but they are a major cause of blindness in developing countries.

Inclusion Conjunctivitis:

Inclusion conjunctivitis is the typical "sexual" chlamydial infection of the eye that you're most likely to see here in the US. Patients present with a chronic conjunctivitis that has persisted for more than three weeks. As in other bacterial infections, the patient will have injection of the conjunctiva and purulent discharge. They may also show follicular "cobblestoning" that develops on the inner eyelids.

This infection occurs mainly in newborns or sexually active teens with a concurrent genital infection. Migration of the bacteria to the eye occurs from hand-eye transmission, and can also spread person to person from shared cosmetics or from improperly chlorinated hot tubs. Newborns can also be infected while passing through the birth tract. The bacteria can be detected with a chlamydial immunofluorescence test or by culture of the conjunctiva. A Giemsa stain will show the classic basophilic inclusion bodies within epithelial cells.

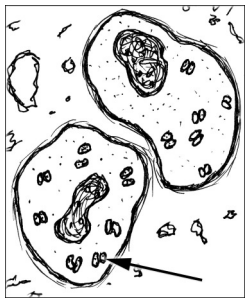
Therapy involves topical antibiotics. Because the bacteria is usually contracted sexually, eyedrops alone won't address the entire problem, so oral azithromycin is also given. Sexual partners also need to be treated.

Trachoma:

Trachoma is the "non-sexual" chlamydial infection of the eye and is caused by a different serotype. It occurs in undeveloped countries with poor sanitation, and outside of the US, trachoma is the leading cause of blindness. The chlamydia bacteria is spread through contact with family members, and can also spread within communities by flies and gnats. The disease causes eventual scarring of the inner surface of the upper eyelid … eventual leading to significant corneal irritation and scarring.

The disease creates a long-lasting, chronic follicular conjunctivitis that eventually progresses to scarring of the eyelids. This scarring can close off the lacrimal gland pores and lead to chronic dry eyes. Scarring can cause the eyelids to rotate inward (entropion), and change the direction of eyelash growth - a condition called trichiasis. Constant rubbing of the lashes against the cornea leads to corneal scarring and eventually to blindness.

Gonococcal Infection:



While gonococcal infection is much rarer than chlamydial infection, it is more serious, as gonorrhea progresses rapidly and can perforate the cornea within a day. These patients will present with redness of the conjunctiva and profuse mucopurulent discharge. This is a serious infection, as the organisms can penetrate through a healthy corneal membrane and perforate the cornea within 24-48 hours, leading to endophthalmitis and loss of the eye. The eye can also act as an entry portal for developing meningitis and septicemia.

With any severe and profuse exudate you should get scrapings and run a culture. A Gram's stain will reveal the hallmark gram-negative diplococci inside infected cells.

Picture: Intra-cellular gram-negative diplococci … usually inside of WBCs

Because the infection advances so rapidly, treatment requires systemic antibiotics with a drug like ceftriaxone (because of the increasing numbers of penicillin-resistant organisms). Topical antibiotics can act as an adjunct but this doesn't work well as the diffuse tearing washes the antibiotic away. If there is severe corneal involvement, or you are worried about your patient's compliance, you may need to admit them so they can be followed more closely.

Babies can contract gonococcal infection during birth -- this is why most states require that they receive prophylactic silver nitrate or erythromycin ointment after birth. We use erythromycin here because silver is irritating and creates a temporary "chemical conjunctivitis."

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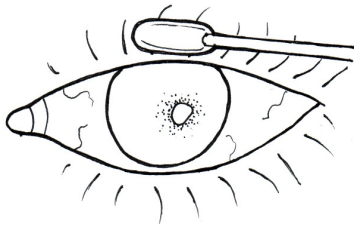
Corneal Abrasions and Ulcers:



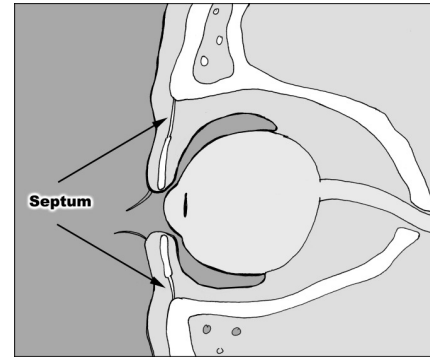
Corneal abrasions are very common and is the most common ER consult we get. Superficial epithelial defects can occur after trauma, infections, or dehydration from exposure. They are very painful, and patients will often have photophobia (pain with bright lights). Fortunately, with aggressive lubrication the superficial epithelial layer heals quickly … literally within a day or two, and the patient feels better.

If an epithelial defect has an associated bacterial infiltrate, this is called a *corneal ulcer*. Ulcers are treated aggressively with antibiotics and should be followed on a daily basis until the epithelial defect has closed. For straightforward, small ulcers, we typically use erythromycin. If the ulcer is large, centrally located, or not healing, then we culture and tailor antibiotics accordingly.

Pimp Alert!!
Contact lens wearers are more likely to have a dangerous infection with *pseudomonas*. In these patients, we cover with ciprofloxacin. If the ulcer looks bad, we'll admit for q1 hour fortified antibiotics (ex. vancomycin and amikacin). Also, we treat any dirty ulcer (i.e., caused by tree branch, fingernail, soil) with more aggressive antibiotics.

With sterile epithelial defects, you can patch the eye to promote lubrication and speed healing. However, you *never* want to patch an eye with a potential infection and you should see patched eyes on a daily basis to make sure an perforating ulcer isn't brewing under that patch.

Pre and Post-septal Cellulitis:



Patients may present with a swollen eyelid that appears to be infected (swelling, erythema, warmth, systemic fever). When approaching cellulites of the eyelid you must determine whether the infection is located pre- or post-septally.

The "septum" is a layer of connective tissue that runs from the lid tarsal plates to the bone of the orbital rim. Infections superficial to this septum can look bad, but generally do well. However, if an infection tracks back *behind* the septum, you're in trouble … and will need to admit the patient for IV antibiotics. These post-septal infections occur commonly from sinus infections that erode through the ethmoid bone, especially in kids.

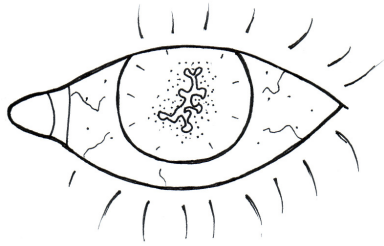
Symptoms of post-septal orbital involvement (a.k.a. orbital cellulites) are pretty obvious: intraorbital soft-tissue swelling will cause proptosis and chemosis (swelling of the conjunctiva). The intraocular muscles can become inflamed producing decreased motility and pain with eye-movement. If the optic nerve is affected they'll have decreased vision and possibly an APD.

Therefore, whenever you see a big swollen eyelid you should always check for signs of post-septal involvement – ophthalmology and ENT has to make this distinction frequently in the pediatric emergency room, as kids can develop bad sinus infections that erode through the ethmoid bone into the orbit.

Herpes Simplex Virus:

Herpes infection of the eye is quite common, affecting over half a million people in the U.S. every year. When herpes attacks the cornea, we call this herpetic keratitis.

Herpetic keratitis is caused by HSV Type-1. This is a common virus, and the vast majority of people contract it during childhood. The virus lies dormant in the trigeminal ganglion and can reactivate, causing cold sores in some people. This reactivation can be triggered by fever, trauma, psychological stress, and UV sunlight. The factors leading to occurrence of the disease at the eye is unclear, though it may have something to do with the virus strain or the patient's immune system.



Patients will present with a red, injected eye and complain of pain. The infection almost always occurs in only one eye, though I've seen bilateral cases. Patients may also exhibit the classical vesicular rash near the orbit. When examining these patients with the slit-lamp, you will see the classic **"dendritic ulcer"** that stains brightly under fluorescein. A single infection typically involves only the superficial cornea and doesn't lead to any long-term sequela. Unfortunately, the infection tends to reactivate. With repeat infections, the virus attacks deeper and deeper areas of the cornea, which can create scarring if the corneal stroma is involved. Deep infections also kill the sensory nerves of the cornea. This decreases corneal sensitivity (you can check with a cotton-swab prior to anesthetic) and can give patients the false illusion that they are getting better.

Treatment is aggressive in order to avoid deep penetration of the cornea. Debridement of the area with a cotton-tipped swab may help, and topical antiviral drops like Viroptic are always given. Acyclovir is often given orally, and continued prophylactic oral acyclovir may decrease the rates of recurrent outbreaks. I also treat nearby skin lesions with topical acyclovir … this drug doesn't penetrate well into the skin, but may decrease viral shedding into the eye. Topical steroids *must* be avoided if there are epithelial defects, as steroids increase viral replication and can lead to a terrible geographic ulcer. With significant corneal scarring, these patients may need a corneal transplant to regain sight.

AIDS and the Eye:

These patients often develop eye infections, especially with low CD4+ counts.

> Nearly all AIDS patients develop a condition called **AIDS retinopathy**. It is a relatively benign state, and is common with CD4+ counts below 200. On fundus exam, you'll see cotton-wool spots (which are infarctions of the ganglion nerve layer), microaneurysms and hemorrhaging. The cotton-wool spots are so prevalent that when finding these spots in a healthy patient (without underlying diabetes or hypertension) you should consider HIV testing. The mechanism behind AIDS retinopathy is unclear, but may result from immune complex deposition in the retinal vessel walls. While AIDS

retinopathy doesn't cause vision problems itself, its continued presence may indicate poor HIV control.

The **cytomegalovirus (CMV)** is the most common opportunistic infection of the eye and is the leading cause of blindness in AIDS patients. Most people contract CMV during childhood (I think I caught it during internship, which really sucked), developing a mono-like illness, and then go on to maintain lifelong immunity with viral suppression. However, the virus can reactivate in AIDS patients because of their decreased immune response. CMV typically occurs with CD4+ counts below 50; and the overall prevalence of CMV is rising as better prophylactic treatment for other deadly infections have allowed more AIDS patients to survive with very low CD4+ counts.

CMV typically attacks the retina, and creates a necrotizing retinitis. Fundus exam shows peripheral areas of white retinal necrosis and associated hemorrhaging. The infection is treated with antivirals like gancyclovir or foscarnet. These drugs are only virostatic, though -- they will suppress the infection, but won't eradicate the virus from the eye. Thus, antiviral treatment needs to be maintained to avoid reactivation. The antivirals can be given by IV (you will likely need to admit the patient for gancyclovir induction) with long-term oral maintenance. Also, after induction a gancyclovir implant can be placed inside the eye itself to allow a slow depot release of the drug.

AIDS patients are susceptible to many other eye infections, including herpes simplex of the retina, toxoplasma, zoster, and syphilis. These are beyond the scope of this book, though.

Famous Quotes

Go away. I'm all right.

Last words of H.G. Wells

Endophthalmitis

Endophthalmitis describes an infection *inside* the eye and is the dreaded complication we fear after eye surgery. Endophthalmitis is really serious … as the eye contains delicate structures and is constructed as a large cavity that can quickly turn into an abscess (eyeball filled with pus).
Endophthalmitis can occur for many reasons … after trauma that exposes the eye to outside flora, or sometimes years after an eye surgery. It can also occur from endogenous infections elsewhere in the body.

Patients present with pain and decreased vision. The infection is easy to spot as these eyes have lots of cell, flare, hazy medium, and you often can't view the retina. The anterior chamber inflammation may be so bad that a layer of pus (called a hypopion) forms along the bottom of the anterior chamber.

Treatment of these patients depends upon their vision … if they see hand-motion or better, we'll do a "tap and inject." This is where we put a needle into the eye to draw out culture and inject broad-spectrum antibiotics. If the vision is "light perception" or worse, then we take the patients to surgery for a vitrectomy to clean the eye out. Visual prognosis is universally poor.

Conclusion:

We could discuss many more eye infections, but these are the important ones to know for the wards and boards. Some of these infections, like blepharitis and corneal ulcers, are very common and you will see these almost daily in the ophtho clinic. Others, like gonococcal keratitis and post-surgical endophthalmitis, are rarer, but important to recognize because of their devastating effects if not treated early.

1. A patient comes into your office in great distress because they have a spot of hemorrhaging under the conjunctiva. Is this a problem and should they be worried?

A few drops of blood spread under the conjunctiva can be alarming, as it looks impressive. Subconjunctival hemorrhage occurs when a conjunctival blood vessel "pops," usually after a valsalva or when bending over. This is generally not a problem, though, as the blood will go away in a few weeks. If the hemorrhage is recurrent, though, start thinking about bleeding disorders. This happened to me once during college after a righteous night of partying and retching – the eye gets really red!

2. What bug do you worry about with contact lens wearers? What antibiotic would you use in a small corneal ulcer in a contact lens wearer?

While most small ulcers can be treated with erythromycin, you must worry about pseudomonas in contact lens wearers. Treat all CL wearers with ciprofloxacin.

3. Can you patch an eye to promote heaing and comfort? Are there situations where you'd avoid patching?
You can patch an eye with an epithelial defect … it feels better and may speed up surface healing. However, you definitely don't want to patch the eye if there is any infection … or chance of infection. Thus, you shouldn't patch anyone with bacterial infiltrate, contact lens, or trauma by "dirty material" like vegetable matter, animal, or dirt. Personally, I only patch eyes after surgery.

4. What are the three kinds of conjunctivitis? How do you differentiate them on history and physical exam?

The cause of conjunctivitis is not always obvious. Generally you'll see the following classic findings:

Viral	watery, follicles, enlarged nodes
Bacterial	mucous
Allergic	itchy and bilateral

5. What's the most common cause of conjunctivitis? How do you treat it?

Viral adenovirus – one of the common cause of colds (rhinovirus actually causes the majority of colds). You treat these supportively with cool compresses, Tylenol, and chicken soup. Warn the patient that they are contagious and encourage them to wash their hands, don't share towels, and throw out their makeup.

6. What's our favorite diagnosis in the eye-clinic (good for explaining chronic irritated, grainy-feeling eyes with stinging and occasional watering). How do you treat it?
This sounds like blepharitis … probably the most common diagnosis in the walk-in clinic. You treat with artificial tears, warm compresses, and lid scrubs. If this doesn't seem to be working, you can try oral doxycycline (don't use in kids, pregnant women).

7. What's a chalazion, stye, and hordeolum? How do you treat them?
A chalazion is a non-infectious inflammation of the meibomian gland … see the anatomy chapter. A stye is like a pimple at the lid margin, usually at the base of an eyelash. A "hordeolum" is a general term that describes an "inflamed gland." It is debatable what this means, so I don't like to use the term myself, but you may run across it.

8. What are the signs/symptoms of herpetic keratitis? How do you treat?
Painful eye with the classic dendritic ulcer. You treat with topical antiviral drops like Viroptic and oral acyclovir.

9. You suspect a patient of having a herpetic infection, based off the shape of her epithelial defect, and you are concerned about corneal scarring. Should you use a steroid to decrease inflammation and resulting scarring?
You should NEVER use a steroid drop in herpetic disease if there is still an epithelial defect, as this will cause a terrible "geographic ulcer." You use topical antivirals like Viroptic and oral acyclovir.

10. Are eyes with herpetic keratitis more or less sensitive to touch?
These eyes are less sensitive to touch as the virus kills the corneal nerves. When HSV is suspected, we check corneal sensitivity with a q-tip or a monofilament prior to anesthetic. Eye sensitivity is an important component of the blink reflex.

11. What is Hutchinson Sign?
You see this with zoster infections of the eye. If you see vesicles at the tip of the nose, this increases your suspicion for ocular involvement as they share the same nerve distribution (the tip of the nose is supplied by the nasociliary nerve branch of V1).

12. What is Reiter's syndrome?
Can't see, can't pee, can't climb a tree. Typically set off by a chlamydia infection and is associated with HLA-B27. These patients often have a conjunctivitis.

13. What's the difference between a corneal abrasion and a corneal ulcer?

A corneal ulcer is an abrasion PLUS infection infiltrate. Ulcers require antibiotic coverage and possible culturing depending upon the severity, size, and location of the lesion.

14. A 21 y.o. man presents with grossly swollen eyelid – a few days before he had a pimple that his girlfriend popped with some nail clippers. Since then his eyelid has swollen up, with redness, mild warmth and tenderness to touch. What specific findings would make you concerned for deeper involvement.

This patient sound like he has an infection of the eyelid. The question is whether it has any post-septum involvement (ie. orbital cellulites). You need to check vision, proptosis, chemosis, decreased eye motion, and pain with EOM. These findings would suggest a dangerous orbital infection and the need for admission, imaging, etc..

Chapter Seven

Neurology

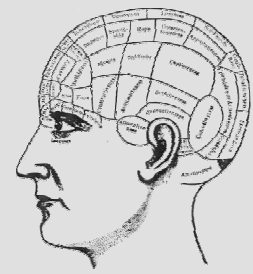
Neurology

by Tim Root
(last updated 9-24-06)

Introduction:

A third of the brain is devoted to the visual system, so neurology is an important topic! While the rest of ophthalmology involves image recognition (your ability to recognize corneal abrasions, disk cupping, and infections under the microscope), neuroophthalmology requires a more cerebral analysis. This makes learning the subject painful at first - trust me, it becomes more entertaining as you progress! For our purposes, I'm going to keep things simple and only cover topics that you should know as a medical student.

Fun Fact!



Phrenology is the study of the morphology of the skull, and was developed by Franz Josef Gall (1758 – 1828). Gall felt there was a direct link between the morphology of the skull and human character and intelligence. While complete bunk, Gall was one of the first to consider the brain the source of all mental activities.

Phrenology was very popular in America throughout the 1800's and its influence can still be seen in our language. For example, people with "high brows" were considered more intelligent than those with 'low brows."

Diplopia:

A common complaint you're going to be faced with is "double vision." Patients frequently complain of doubling … and sometimes they actually mean it! Often, however, they just mean that their vision looks blurry. Technically, the phrase diplopia describes the symptom of seeing two different images of the same object, and that's what we are going to discuss!

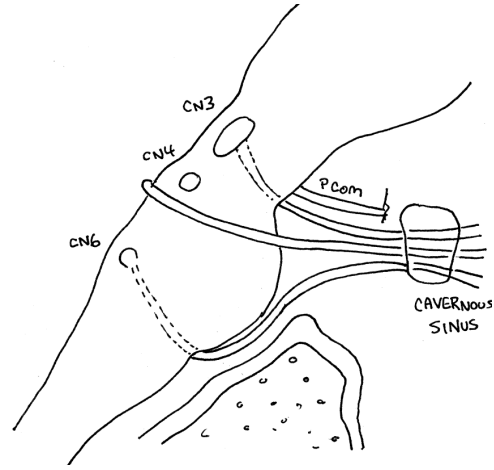
When faced with a diplopic patient, there is an important question you must immediately answer: before breaking out your arsenel of neuroophthalmic flags, prisms, and muscle lights, you must distinguish whether the diplopia is **monocular** or **binocular**. If the double-vision remains when you cover an eye then you have a monocular diplopia. You should breath a sigh of relief at this point - because monocular double-vision isn't a neurologic problem at all and your exam just got easier!

Monocular doubling is often caused by a refractive problem in the front part of the eye. There aren't any mechanisms of doubling that occur at the retina or further back in the neuro pathway. The most common cause of monocular diplopia is astigmatism, an abnormal curvature of the corneal surface. New onset astigmatism could occur from corneal deformation from an overlying lid lesion or after surgery with tight stitches through the cornea. Other causes of monocular diplopia include cataract irregularities, lens displacements, or primary problems with corneal curvature such as keratoconus.

Binocular diplopia, however, occurs when the eyes do not move in sync with each other. This can could occur from **nerve** lesions (a palsy of CN3, CN4, or CN6), extraocular **muscle** abnormalities (such as the muscle-swelling that occurs with Grave's Disease), or derangements at the neuromuscular **junction** (myasthenia gravis). Let's explore the cranial nerve palsies first.

Cranial Nerves and EOMs

Three cranial nerves control the movements of the eyeballs. The relationships of these muscles can be quite complex as the eyeballs are neurologically "yoked" together and every muscle has multiple vectors of force, depending upon the direction that the eye is looking. Picking up a nerve palsy is often challenging, especially if the palsy is only partial.



There are numerous causes for the individual nerve palsies, including microvascular nerve strokes, tumors, and aneurisms. If you're like myself, you're probably not up-to-speed on your neuroanatomy. I've drawn this cartoon picture of the brainstem for you to reference over then next few pages.

Third Nerve Palsy

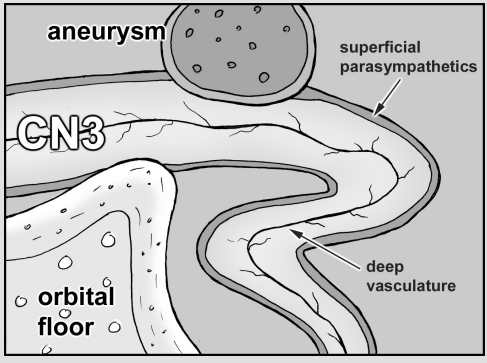
Oculomotor nerve pasly is the easiest to detect because a complete 3rd nerve palsy looks dramatic. The majority of the extraocular muscles are innervated by CN3, so when knocked-out, the eye deviates down and out

because of the still functioning abducens and superior oblique muscles. In addition, the levator palpebrae (the main lid retractor) is innervated by CN3 giving you a severe eyelid ptosis. Finally, the parasympathetic pupil-constrictor fibers from the Edenger-Westphal nucleus travel in CN3, giving you a "blown pupil."

Most third nerve palsies are caused by nerve ischemic events in the diabetic or hypertensive patient. The one thing you really need to worry about in these patients is a compressive aneurysm pushing on the nerve. These aneurysms commonly occur at the junction of the posterior communicating artery and the internal carotid artery. Compressive lesions like this affect the parasympathetic nerve component: a blown pupil is a potential emergency. Whenever you have pupillary involvement, you need an MRI and angiography to rule out a dangerous aneurysm.

Pupil Involvement with CN3:

Occulomotor palsies often have pupillary involvement because the parasympathetic nerves innervating the iris travel with the inferior division of CN3 into the orbit. Pupillary involvement depends upon the cause of the palsy -- compressive lesions tend to involve the pupil, while vascular lesions actually spare it! This picture isn't drawn to scale, but graphilcally demonstrates what I'm talking about.



This occurs because the parasympathetic nerves course along the surface of the oculomotor nerve making them susceptible to compressive lesions from the outside such as an aneurysm from the posterior communicating artery, boney structures, or the uncal portion of the temporal lobe. Ischemic lesions (caused by HTN and diabetes) occur deeper within the oculomotor nerve and thus spare the superficial parasympathetic fibers.

If you have a patient with CN3 loss and pupillary involvement, you should order an MRI and an angiogram to look for the compressive site. If there isn't pupillary involvement, they are probably suffering from a vaso-occlusive problem, so you should check their glucose and blood pressure.

Abducens (VI):

The abducens nerve controls the lateral rectus. Loss of CN6 renders the eye unable to abduct (turn out). Patients will go cross-eyed, so to compensate they may turn their head to the ipsilateral (same) side to avoid double vision.

If you look back in that drawing of the brainstem, you'll see that the abducens nerve is located further down the brainstem, all by its lonesome, in the pons. The nerve emerges even further down at the ponto-medulary junction and has to run up the floor of the skull to get to the cavernous sinus and through that into the orbit. Where the nerve enters the cavernous sinus, it makes an abrupt 90-degree bend. Something about this abrupt turnmakes the 6th nerve susceptible to high intracranial pressure. Patients with high ICP from pseudotumor commonly have their 6th nerve(s) knocked out – an abducens palsy is incorporated into the Dandy criteria for diagnosing PTC.

Fun Fact!

Crocodiles shed tears, but this isn't a sign of grief. These secretions help shed salt-water from the eye. Thus, the term "crocodile tears" is used to describe false tears. In ophthalmology, we use the term to describe aberrant regeneration after 7th nerve injury - nerves that normally control salivation are routed to the lacrimal gland. This makes you "cry" when you see food. You can treat this by injecting botox into the lacrimal gland.

Aberrant regeneration occurs with other cranial nerve palsies as well … the most commonly seen is after a 3rd nerve palsy. As the oculomotor nerves grow back to their muscles they can get mixed up. For example, a patient could look medially (activating their medial rectus) and their eyelid can shoot up (inappropriate co-contraction of the levator palpebrae).

Trochlear Nerve (IV):

The fourth trochlear nerve (CNIV) innervates the superior oblique. The trochlear never is the hardest cranial nerve palsy to diagnose! These patients have an upward deviation of the affected eye and a "cyclotorsion" twisting of the eye that makes them tilt their head away from the lesion. Don't try to memorize these deviations … in a few paragraphs I'll cover the

anatomy of the trochlear muscle which will make is easier to conceptualize these findings.

A trochlear nerve lesion is caused by either trauma, ischemic events (like all the lesions), or are congenital with later decompensation. The fourth cranial nerve is the skinniest nerve and runs the longest distance inside the cranial vault. This long passage makes it more susceptible to injury if the brain sloshes around and bounces the tentorium. The nerve is also suceptable to being pulled from the root where it pops out the back dorsum of the midbrain. The long course also makes it susceptible to neoplasm. If we break it down by cause:

1/3rd Trauma

1/3rd Congenital

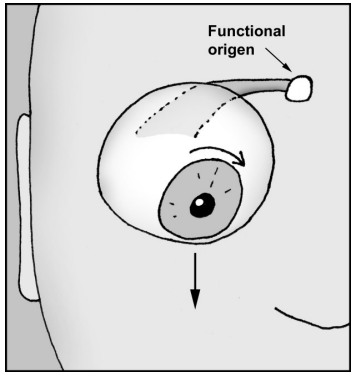
1/3rd Ischemic (diabetic)

1/3rd Tumor

That's a lot of thirds, I know, and they are not exact number. Reports differ depending upon what age-group you look at: certainly more 4th palsies occur in elderly males from trauma and more congenital palsies are found in the pediatric clinic. Ask about history of closed-head injuries and check old photographs to look for an old head-tilt … this would indicate an old/congenital palsy that has recently decompensated.

Troclear Muscle Action:

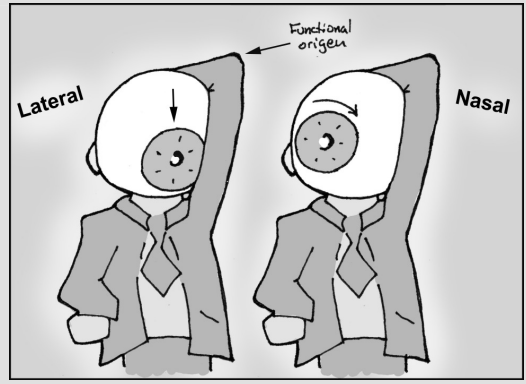
The trochlear muscle runs from the back of the orbit, forward through a trochlear "pully" located next to the upper nasal bridge, before running behind the eye. This pulley system completely changes the force of the superior oblique … you can think of the trochlea as the "functional origen" of the muscle.



As you can see in this picture, the superior oblique muscle grabs onto the back of the eyeball and yanks the eye down. There is also an intorsional component that rotaes the 12-o'clock corneal limbus towards the nose. This explains the head-tilt these patients develop.

A deeper look at the superior oblique:

To simulate the action of the superior oblique, you can pretend that your head is a large eyeball. Throw an arm up and wrap it around the back of your head. Your elbow becomes the trochlear pully … if you pull your arm, you're whole head should twist. The direction of head movement, either up-down or rotational, will depend upon which direction you're looking when you pull.



The same thing occurs in the eyeball … your patient will see vertical diplopia when looking toward the nose (such as when reading a book) and see more rotational doubling when looking to the side. Think about that one for a minute!

Summary of the EOMs:

Beyond the information above, there isn't much to localizing a cranial nerve lesion. Just think about the anatomy: fi a single nerve is affected, then you know it's somewhere along that nerve's tract. If all three nerves are knocked out, then the lesion is probably near the cavernous sinus where the three nerves are bunched together.

The number one reason that any of the cranial nerve gets knocked out is from a vasculitic event, usually from diabetes. Many of these isolated cranial nerve palsies don't need imaging - such as an isolated 6th nerve palsy in an elderly diabetic. However, you don't want to miss an aneurysm or mass lesion, so no one will fault you for over-imaging. Here's the high-yield facts you should know:

CN3: The eyes are "down and out" with droopy eyelid. Think of aneuryms if the pupil is blown.

CN4: Patient tilts their head away from lesion. Think of trauma and congenital head-tilt that has just decompensated with age.

CN6: The patient is "cross-eyed." Consider increased intracranial pressure.

Myasthenia Gravis:

Myasthenia gravis is a rare autoimmune disease in which the body develops autoimmune antibodies to the nicotinic acetylcholine receptors located at the neuromuscular junction of striated muscle. This leads to fatigable muscles and often involves the eye, causing diplopia and ptosis.

MG patients develop autoantibodies that actually bind to the receptor and block the receptor binding sites, and eventually destroy the receptor entirely, leaving patients with decreased numbers of Ach receptors … once the number drops below 30% normal, then the patient becomes symptomatic, with easily fatigability. Interestingly, only striated muscle is affected, as both smooth and cardiac muscle appear to have different antigenicity and are unaffected with this disease. The bulbar muscles, however, are quite susceptible, and the majority of patients with MG have ocular complaints - the ophthalmologist is often the first doctor to diagnose the disorder.

The diplopia and ptosis is usually worse on prolonged upgaze … you can test this by having your patient look at your raised finger to see who tires out first. More definitive diagnosis can be made via the Tensilon test— give edrophonium chloride (an anticholineresterase) and look for improvement in symptoms. We don't actually do this test in our office because of the difficulty of starting IV lines and potential toxic reactions such as sweating, salivation, bronchospasm, and bradycardia. More commonly we'll perform a rest-test or ice-test where you have the patient hold an icepack over their closed eyes and then remove it and look for improvement. The neurologists can also perform EMG studies.

Systemically, these patients can have problems with mastication, talking, drinking and difficulty swallowing. Aspiration pneumonia and respiratory failure from a reduced gag reflex and inability to clear secretions is the big killer with this disease. Remember, if your patient has MG, work them up for a thymoma and check their thyroid levels.

Neuritis and Neuropathies of the Optic Nerve

Personally, I found this topic very confusing because the terms "optic neuritis" and "optic neuropathy" sound very similar. After all, what's the difference between an "itis" and an "opathy?" There's really only three main optic-nerve entities you need to be aware of, and each has a completely different mechanism:

1. ON (Optic Neuritis):
An "inflammation" of the nerve, often demyelinating. The cardinal signs in these patients are decreased vision (especially color vision), pain with eye-movement, enhancement of the optic nerve on MRI, and an association with multiple sclerosis. Occurs in younger patients. I'll go into more detail in a few paragraphs.

3. ION (Ischemic Optic Neuropathy) … sometimes called NAION (non-arteritic ischecmid optic neuropathy)

This is a localized ischemic event at the junction of the optic nerve as it enters the back of the eyeball. This portion of the optic nerve has no elastic "give" and a small vascular insult here can lead to swelling and vision loss … creating a localized compartment syndrome. The hemispheric vascular supply to the optic nerve head usually generates an altitudinal visual defect. This entity usually occurs in middle-aged people with small optic disks (the so-called "disk at risk").

2. GCA (Giant cell arteritis) ie. temporal arteritis

Temporal arteritis occurs as a result of a vasculitis in the medium and small-sized arteriies around the head. The vasculitis can lead to a sudden occlusion of the blood supply to the retina and eye leading to sudden and permanent vision loss This happens in older patients, usually over 70 years of age.

Let's explore each of these entities in more detail …

Multiple Sclerosis and Optic Neuritis:

Multiple sclerosis is a demyelinating disease of the CNS with lesions occurring at "different times and different places." MS is most common in young white women from northern climates and if the lesion hits the optic nerve, then we call this finding optic neuritis. In fact, 90% of patients with multiple sclerosis will develop optic neuritis at some point … and conversely, patients with "optic neuritis" sometimes go on to develop multiple sclerosis. Think about that one for a moment!

<u>Signs and symptoms of optic neuritis include:</u>

- Sudden vision loss (central scotoma is classic)
- Decreased contrast and color sensitivity
- Pain with eye movement
- Optic nerve head edema
- Afferent pupillary defect

A patient with optic neuritis needs an MRI of the brain and orbits to look for enhancing lesions. The more lesions found, the higher the chance of later developing multiple sclerosis. Patient with optic neuritis are treated with IV steroids, which will speed recovery, but won't ultimately affect the outcome of the disease. WARNING: You treat with IV steroids only, as oral steroids actually increase the progression to MS (and will get you sued)! If enhancing lesions are found in the brain, then you can discuss possible treatment with interferons like Avonex to also decrease progression.

Fun Fact!

People blink, on average, once every 5-6 seconds.
Woman blink almost twice as often as men.

Temporal Arteritis:

Temporal arteritis (also known as Giant Cell arteritis) is an important syndrome to keep in the back of your head. While not terrible common, you can save a patient from complete blindness if you treat them appropriately.

This disease process is similar to polymyalgia rheumatica, except that the vasculitis affects arteries around the head. If the blood supply to the eye is shut down, then they can have catastrophic vision loss. These patients are almost always OLD (over 60 and more commonly over 80 years of age) and present with sudden, painless vision loss. Other preciding systemic symptoms (these are pathognomonic symptoms, so memorize them) include:

- Scalp tenderness and headache
- Jaw claudication
- Polymyalgias
- Fevers, night sweats, weight loss

If you suspect GCA, get a stat ESR and CRP, as these are sensitive markers of inflammation. Normal ESR is approximately half the patient's age (i.e., an 80 y.o. man can have ESR up to 40). Unfortunately, these labs aren't very specific and more definitive diagnosis is made via a temporal artery biopsy (dissect out the artery at the temple and send it to pathology). On pathology you find disruption of the internal elastic lamina and sometimes giant cells (the presence of these cells isn't actually necessary for the diagnosis).

You treat temporal arteritis with steroids to decrease inflammation. The steroids won't often help with the lost vision, but will decrease the potential for loss in the remaining eye, which can be affected in days. Unfortunately, steroids also decrease diagnostic yield on your biopsy … which places you in a diagnostic dilemma: do you hold off steroids until after the biopsy, or start

steroids and potentially mess up your biopsy results? Well, the answer is that you start the steroids *immediately* to keep the other eye from being affected and blinding the patient. The biopsy can be delayed for up to two weeks and still be ok, despite the steroids.

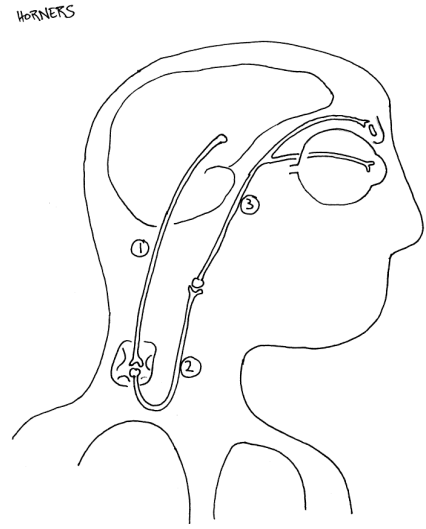
The Pupil

Horners Syndrome

Iris movement is controlled by the parasympathetic (constriction) and sympathetic (dilation) systems Horners occurs when the sympathetic pathway gets knocked out … leading to pupil constriction, often associated with mild lid ptosis and anhydrosis (decreased sweating) on that side. As you can see in the picture to the right, the sympathetic chain is long and complex, and can be affected at any level. To localize the lesion we use a series of eyedrops:

Cocaine Test

The first test we perform is the cocaine test … just to decide if this patient REALLY has a Horners pupil or not. Cocaine stimulates sympathetics by decreasing norepinephrine uptake at the synaptic cleft. If a patient (a Horner's patient) has no sympathetic tone inside the eye, then cocaine won't build up norepinephrine and thus has no effect on that eye. However, the good eye will dilate like the dickens! It's not always easy to obtain cocaine in private practice, but you should be able to get some in most hospitals as ENT people use it to control nose bleeding in surgery.



Paradrine Test

Now that we know there's a sympathetic palsy, we need to localize the lesion … just like the muscles throughout the rest of the body, there are three neurons in the pathway from the brain. Unlike a leg muscle, we can't check the pupillary dilation reflex by hitting it with a hammer … but we can stimulate that final 3rd order nerve by pharmacologically hammering it with hydroxyamphetamine. If that pupil still won't dilate, then you now the final "lower motor neuron" is dead. If the pupil DOES dilate, then you have a

"higher order" nerve that's out - lots of bad things can occur along this upper-pathway (carotid dissections, pancoast tumors, etc.) so procede to imaging.

Horners syndrome tests is a hard topic to cover, and you'll find it difficult to really remember those drops and their purpose until you see your first Horners patient. One catch phrase you should remember, though: if a patient complains of a "painful Horners" think of a carotid dissection and move quick.

Adies "Tonic" Pupil

An Adies pupil is the opposite of a Horners … in this case the *parasympathetic* (constrictor) pathway gets knocked out on its way to the iris sphinter muscles. On exam, the eye is dilated and doesn't constrict to light (we're blocking the parasympathetic pathway from the Edenger Westphal). The pupil will constrict with near vision- but very very slowely. That's why we call it a "tonic pupil," it's tonically slow.

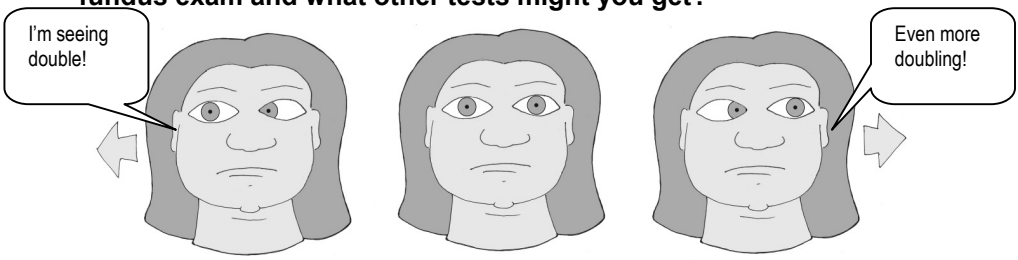
Fortunately, for us, the parasympathetic pathway is much shorter than that convoluted sympathetic pathway, and so it's more benign. The parasympathetic plexus sits right behind the eye and commonly gets knocked out after a viral infection.

1. You have a patient with diplopia. His left eye is turned down and out and his lid is ptotic on that side. What nerve do you suspect and what should you check next?
This sounds like a CN3 palsy, and you should check his pupillary reflex. Pupillary involvement means the lesion is from a compressive source such as an aneurysm.

2. Why do diabetic patients with oculomotor paralysis have "sparing of their pupil?"

Interestingly, with CN 3 palsies caused by vascular problems the pupil is typically spared. This occurs because the parasympathetic pupillary fibers run along the surface of the nerve … which makes them susceptible to aneurysm/tumor compression but resistant to deeper infarction.

3. This 32 year old overweight woman complains of several months of headaches, nausea, and now double vision. What cranial nerve lesion do you see in this drawing. What other findings might you expect on fundus exam and what other tests might you get?



This looks like an abducens palsy … actually a bilateral 6th nerve palsy as the patient can't get either eye to move laterally. While the majority of abducens palsies occur secondary to ischemic events from diabetes, this seems unlikely in a young patient. Her symptoms sound suspicious for pseudotumor (obese, headaches). You should like for papilledema of the optic nerve, get imaging, and possibly send her to neurology for a lumbar puncture with opening pressure.

4. A patient is sent to your neurology clinic with a complaint of double vision. Other than trace cataract changes, the exam seems remarkable normal with good extraocular muscle movement. On covering the left eye with your hand, the doubling remains in the right eye. What do you think is causing this case of diplopia?

The first question you must answer with a case of diplopia is whether it's monocular or binocular. This patient has a monocular diplopia. After grumbing to yourself about this patient being inappropriately referred to your neurologic clinic, you should look for refractive problems in the tear film, cornea, lens, etc..

5**. A patient complains of intermittent double vision that seems to be worse in the evenings. On exam you find a confusing diplopia that doesn't seem to map out to any particular nerve palsy. What else is on your differential as a cause, and what tests might you perform in the office?**

Myastenia gravis and thyroid orbitopathy are both great masqueraders and cause diplopia . The double vision in myastenia patients can look like an isolated nerve palsy, a mixture of nerves, or may not fall into any specific nerve combination. A changing palsy is more indiciative of a process like MG. You can check for fatiguable ptosis by prolong upgaze (hold your arm up and see who gets tired first). In addition, you can perform a cold-pack test, or possibly a Tensilon test.

6. You are giving a tensilon test to a suspected MG patient and he collapses. What do you do?

Your patient may have a reaction to the anticholinersterase such as bradycardia or asystole. You should have a crash-cart handy and administer atropine. Hopefully, this scenario never happens to you.

7. A patient with diplopia is finally diagnosed with myasthenia gravis after a positive ice-pack test and a positive acetylcholine receptor antibody test. What else should you work up this patient for.

You should check for a thymoma, which is highly associated with MG. Also, check their thyroid level as 20% of myasthenia patients also have Grave's disease.

8. A 26 year old woman presents with decreased vision in her left eye that has gotten progressively worse over the past week. The eyes seems to ache and the vision worsens with exercise. On exam she is found to have 20/200 vision, trace APD, and markedly decreased color vision in the affected eye. The optic nerve is mildly swollen on that side. What does this patient most likely have?

This patients age, color vision, and progression are all classic symptoms of optic neuritis. She also describes the classic Uthoff symptom of worsening symptoms with body-temperature (excersize or shower). Many of these patients describe minor pain with eye-movement: the optic nerve is inflamed and any tugging on the nerve (moving the eye) is going to irritate it.

9. A patient develops optic neuritis. Should you treat with steroids? Do you give your patient IV or oral steroids? Will the MRI findings of additional demyelinating lesions change your management? Do you tell the patient that she will develop MS?

The ONTT study has shown that steroids can speed recovery from optic neuritis, but have little effect on long-term outcome. Suprisingly, the study also showed that oral prednisone may actually increase reoccurrence of optic neuritis. You can give IV Solu-Medrol but DON'T give oral prednisone!

The presence of optic neuritis does not necessarily mean the patient will develop multiple sclerosis, especially in the setting of a negative MRI. The patients long-term risk for developing Multiple Sclerosis depends upon the number of CNS lesions found on presentation. IF there are no CNS lesions, then the future risk is only 15%. This jumps up to 50% or more with 3+ lesions. In these patients, you can discuss treatment with Avonex.

10. A 84 y.o. man was out golfing with his buddies and developed vision loss in his right eye. He has no past ocular history, no medical problems. No complaints of flashes or floaters, just that things "look dimmer" in his right eye. What other questions should you ask about his symptoms.

There are many questions you should ask … but with any elderly person with vision loss, be sure to ask about the symptoms of temporal arteritis. Specifically, scalp tenderness, jaw claudication, and polymyalgias (muscle aches in multiple places).

11. The patient admits to "not feeling good" and "it hurts my head to brush my hair on the right side" for the past week but denies all other symptoms. Should you order any labs? Start any medications?

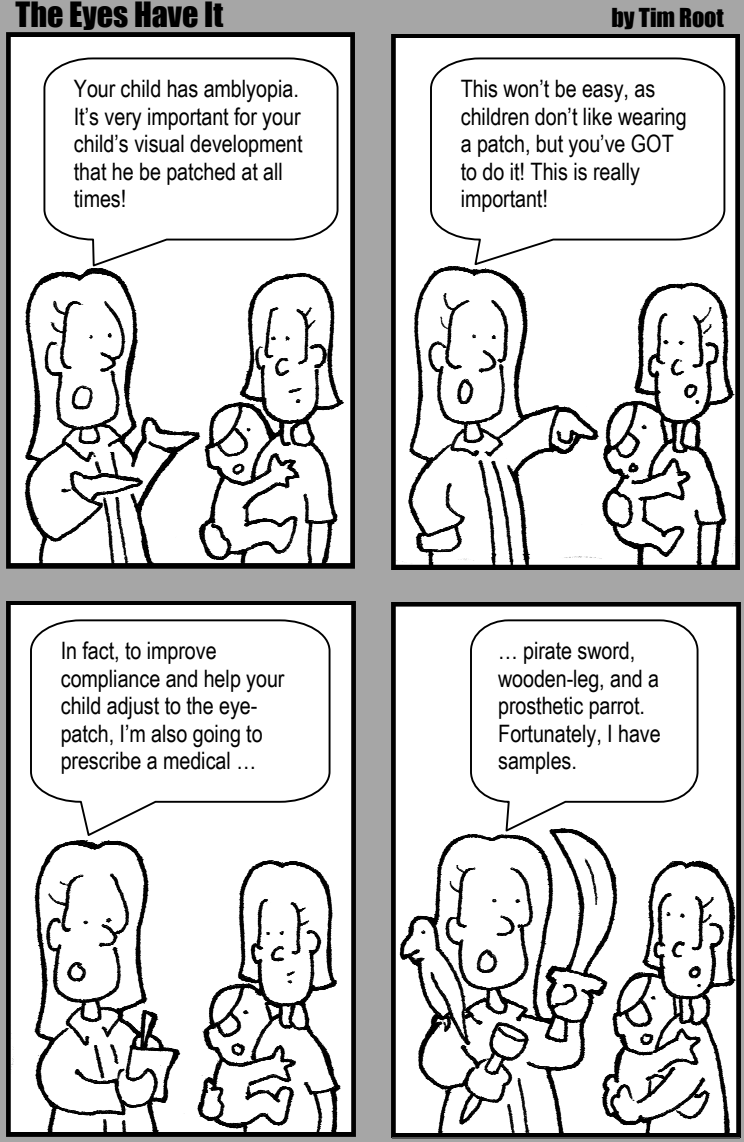
If you have any suspicion for GCA, you pretty much *have* to order a ESR and CRP. Start oral prednisone (about 1mg/kg/day) immediately and set up for temporal artery biopsy within a week or so. Steroids won't help much with his lost vision, but decreases the risk to the other eye, which can be affected within hours to days.

12. A young man complains of complete vision loss (no light perception) in one eye, however, he has no pupil defect. Is this possible? How might you check whether this patient is "faking it?"

Assuming the rest of the eye exam is normal (i.e. the eye isn't filled with blood or other media opacity) this patient should have an afferent pupil defect if he can't see light. There are many tests to check for mali**n**gering: you can try eliciting a reflexive blink by moving your fingers near the eye. One of my favorite techniques is to hold a mirror in front of the eye. A seeing eye will fixate on an object in the mirror. Gentle movement of the mirror will result in a synchronous ocular movement as the eye unconsciously tracks the object in the mirror.

Chapter Eight

Pediatrics



Pediatrics

by Tim Root
(last updated 6-27-06)

Pediatrics ophthalmology is a large field and a difficult one as it's really hard to check the eyes of an uncooperative child. Since I don't yet have a good grip on the subject, I'm only going to mention a couple of important topics and concepts.

Famous Quotes

Children today are tyrants. They contradict their parents, gobble their food, and tyrannize their teachers.

Socrates (470-399 B.C.)

Amblyopia (a.k.a. "lazy eye"):
Amblyopia, is decreased vision in an eye because of disuse of the eye during childhood development. The prevalence in the USA is 2-5%, the major risk factors being prematurity, developmental delay, and a family history.

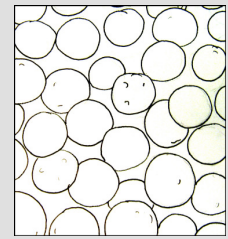
The visual system is a plastic system that continues to develop during childhood until around 6-9 years of age. During this time, the wiring between the retina and visual cortex is still developing. Any visual problem during this critical period, such as a refractive error or strabismus (cross-eye) can mess up this developmental wiring ... resulting in permanent visual loss that can't be fixed by **any** corrective means when they are older.

Competitive Wiring!
If you don't use an eye, the nerve fibers from that eye don't develop ... in fact, the eyes are in **competition** with each other. The afferent nerve connections of the strong eye become numerous while the weak (unused eye) atrophy and decrease in number. In animal studies, the occlusion of one eye leads to loss and atrophy of cells in the LGN (lateral geniculate nucleus).

Fortunately, the situation can be reversed by giving the bad eye a competitive advantage. Weakening the strong eye (i.e., **penalizing** it) with the use of patches, eyedrops that blur vision, and glasses with filters, gives the weak eye time to re-grow its afferent nerve connections.

How to Detect:
It's important to pick up this when young when it can be corrected, as once a child reaches 7-10, they can't be helped at all. With very young children (ones who can't talk) try covering each of the eyes with your hand ... if the child is more irritated when a particular eye is covered, he/she may not be seeing well out of the other eye. In older children, you can check corrected vision. Early correction is important!

Fun Fact!



The word lens is named after the lentil plant (greek name *Lens culinaris*) whose 2 – 9 mm disk-shaped seeds bear a remarkable resemblance in shape and size to the human lens.

The lentil legume was one of the first agricultural crops and was grown over 8,000 years ago. Introduced in the U.S. during the early 1900's, the "lentil bean" is grown in drier Washington, Idaho, and Western Canada and the seeds can be used in soups, stews, casseroles and salad dishes.

Strabismus:

Strabismus describes when the eyes are not aligned ... such as when an eye is turned in (cross-eyed or esotropic) or turned out (wall-eyed or exotropic). Here are some terms we use in ophthalmology to describe misalignments:

Esotropia (ET): The eyes are turned inwards (cross-eyed) all the time

Exotropia (XT): The eyes are turned outwards (wall-eyed) all the time

Eso/Exo-phoria: When the eyes are only occasionally misaligned, usually under conditions of stress, illness, fatigue.

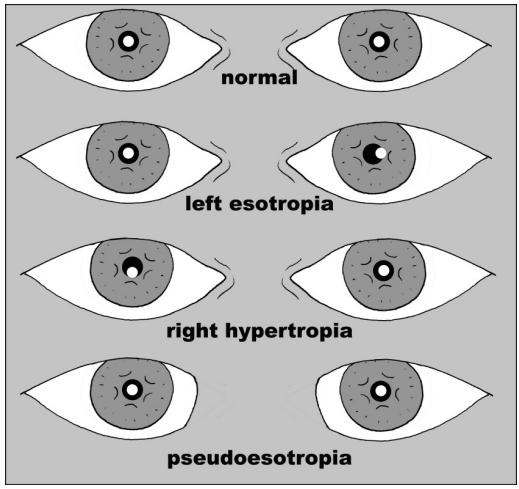
You can pick up phorias with the cross-cover test. Since the cross-cover test breaks binocular vision, the phoric eye will wander off axis when it has nothing to focus on.

Strabismus as a major cause of amblyopia

If a child has misaligned eyes, they can unconsciously suppress one of their eyes -- this helps them to avoid seeing double. However, this kind of disuse leads to amblyopia and permanent visual loss. Adults don't have this ability

… if they develop strabismus (such as from a nerve palsy or trauma) they are unable to suppress their eyes, and will see double.

Detecting Strabismus in babies



A quick way to determine alignment in babies is by shining a penlight into their eyes. You can determine alignment by looking at the corneal-pupil light reflex (the light reflection off the cornea) to make sure it is centered over the pupils of each eye. A large percentage of newborn infants will have some tropia at birth … this goes away in a few months.

Don't be fooled by **pseudostrabismus** – this is the illusion of cross-eyes caused by nasal epicanthal skin folds (found in many Asian and young children) or close inter-pupillary distance. When looking at these children, less white sclera is seen nasally and the child "looks cross-eyed," but will have normal corneal light reflexes. Children outgrow these epicanthal folds as the bridge of the nose becomes more prominent.

You can measure strabismus with prisms. With younger children, you can use the **Hirschberg's test** – a quick method to estimate the prism deviation. Basically, for every mm the corneal light reflex is off, equals approx. 15 diopters of prism.

Treatment:

Make sure that the child isn't suffering from a refractive error and treat the amblyopia … many cases of strabismus will improve or resolve by just doing these things. Surgery can also be done … consisting of shortening muscles with resection and loosening other muscles. The goal of strabismus surgery is correct alignment when looking straight ahead … it doesn't fix the underlying muscle problem (the patient isn't going to have perfect alignment in all eye positions).

Famous Quotes

My mother had a great deal of trouble with me, but I think she enjoyed it.

Mark Twain

Shaken Baby Syndrome:

This is a depressing topic (I won't be adding any funny cartoons to this section). This terrible condition is important to recognize, as it is preventable.

New babies cry a lot … on average three hours a day (and often for no apparent reason). Some babies cry significantly more than this! Frustrated caregivers may pick up the child and shake it if suitably irate. This shaking can become a reinforcing response as the infant becomes somnolent afterwards and stops crying (exactly the response the caregiver was hoping for). Biological fathers and boyfriends are the most common perpetrators, but anyone can be the attacker – this phenomenon spares no ethnicity, religion, culture, or social class.

Infants are predisposed to physical damage from shaking as they have big heads and weak neck muscles. As the head whiplashes back and forth, the acceleration and deceleration forces traumatize the brain in a big way …

Presentation:
These children are typically 5 to 10 months of age and present with somnolence, seizures or coma. The classic triad of exam findings include:

1. **Intracranial hemorrhage**: usually a subdural hemorrhage secondary to tearing of the small bridging veins between the dura mater and arachnoid.
2. **Brain swelling**: From shearing forces, diffuse axonal damage, and secondary edema and infarction.
3. **Retinal hemorrhages**: specific findings (see below)

The child can also have other physical findings such as bruising of the body trunk (where the shaker grips the child) and fractures of the skull, long bones, or ribs. Remember: be suspicious for abuse when you see fractures at different stages of healing.

Retinal Findings:
An ophthalmologist needs to be called in for any case of potential shaken baby to evaluate the retina. These kids have specific retinal hemorrhages that aren't really seen in any other condition:

1. Large retinal hemorrhage located in many quadrants of the eye, located in *all* layers of the retina (subretinal, intraretinal, and preretinal)

2. Retinoschisis cavities. A **schisis** is a split between layers of the retina, and is almost pathognomonic for shaken babies.

It's imperative to write a descriptive note in the chart (we usually have the attending write this note for litigation reasons) and document the bleeding with fundus photos. You probably want to take these photos soon, as hemorrhages can resolve in only a few days!

What about other causes of retinal bleeding?

Studies have found that household injuries, such as a fall from caregiver's arms or furniture doesn't usually cause significant retinal hemorrhages (even with significant brain injury). Birth trauma often causes retinal hemorrhages, but these are usually limited to dot-blot or flame hemorrhages and resolve in the first few months. CPR with chest compressions rarely causes significant hemorrhaging. The hemorrhages in SBS are impressive and similar retinal bleeding isn't seen except with big traumas like a high-speed car wreck or a multi-story fall.

Be sure to look for coagulopathy with basic lab testing … including CBC, coags, platelet count and bleeding time.

Prognosis:
While a third of these kids have no long term sequela, the long-term prognosis is generally bad. 20% of the kids die outright and the remaining kids end up with mental retardation, developmental problems, blindness, paralysis, and behaviour changes.

Famous Quotes

Whatever their other contributions to our society, lawyers could be an important source of protein.

Guindon cartoon caption

The Leukocoric pupil:

Every newborn receives a baseline exam by their pediatrician, and part of this exam involves checking for a good red-retinal reflex. Leukocoria describes a white-colored pupil, and this finding is one of concern and demands an ophthalmology consult as the causes may be serious. Potential causes that you should be aware of include:

1. Cataract
2. Retinoblastoma
3. ROP (retinopathy of prematurity)

Many different medical problems, such as persistant hyperplastic primary vitreous, can also create a white pupil … but let focus on these three:

Congenital Cataract

A cataract in a newborn can occur from many sources. They can be idiopathic, genetic, metabolic disorders, child abuse trauma, or caused by one of the maternal TORCH infections during fetal development. Whatever the cause, it is important to remove a cataract in these children as soon as possible, as they are amblyogenic and will lead to permanent vision loss. Replacing a lens is tricky, however, as babies are tiny, generate impressive inflammatory responses, and their eye "prescription" is still changing as the eye continues to grow until age 2. Thus, the cataract is removed as soon as possible, and a lens can be placed at a later date … parents have to deal with contact lens or thick glasses in the meantime.

Retinoblastoma

Retinoblastoma is a tumor of the primitive retinal photoreceptors. The tumor grows on the retina and forms a white or cream-colored mass that can completely fill the eye, creating a white iris, and often a retinal detachment.

Retinoblastoma is the most common malignant ocular tumor in children. That being said, the cancer is still very rare, with only 250-500 new cases reported in the United States each year. You don't want to miss this one, though, as failure to diagnose RB results in the death of a child.

These children are under 4 years of age, with the average age of diagnosis 18 months. There are different types of RB, and the tumor can arise from a random somatic mutation or develop from several germline inheritance patterns.

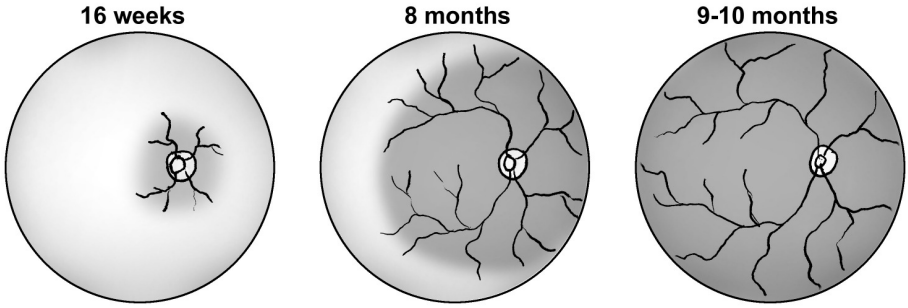
Treatment modalities are many, but decisive treatment involves enucleation (removal) of the entire eye to avoid seeding tumor cells into the orbit. The tumor usually spreads by extension down the optic nerve toward the brain so it is important to get a good optic nerve section upon removal with careful microscopic evaluation for margin involvement. The overall survival rate in the US is very good, approaching 90% … but only if the tumor is recognized early.

Retinopathy of Prematurity

ROP is an important disease process that we deal with on a weekly basis in our pediatric/retina service. We check many premature infants in the NICU

and every week we laser ROP eyes or take kids to surgery for vitrectomy or retinal detachment repair. Here's how ROP works:

The retinal blood supply begins formation around week 16 of gestation, with retinal vessels springing forth from the optic disk and spreading outwards in an expanding fan toward the edges of the retina. By the 8th month this vasculature has reached the nasal retinal-edge, and within a few more months the whole retinal blood supply has formed 360 degrees.



Everything sounds good, right? Well, the problem occurs if a kid is born premature. When this happens, the areas of peripheral retina that haven't yet developed blood supply can become ischemic and produce VEGF with resulting neovascularization. This neovascularization can bleed, create traction, and produce retinal detachments. A leukocoric "white pupil" can result if the retinal detachment is big enough. The more premature a child is born, the more this unfortunate sequence of events is likely to occur.

We treat these kids in a similar fashion as diabetic retinopathy … by killing off the peripheral iscemic retina with laser burns or cold-cryotherapy in an attempt to shut down VEGF production.

Pimp Questions

1. What is amblyopia. What causes it?
Poor vision in an eye without any apparent external or refractive explanation. Caused by disuse of the eye at an early age, usually secondary to strabismus or an unrecognized refractive error at young age.

2. How do you treat amblyopia?
You treat by "penalizing" the good eye. Using a patch over the good eye forces the amblyopic eye to work. Also, you need to treat any cause of the amblyopia such as refractive errors.

3. You suspect a baby of having strabismus, but because of the baby's age, you aren't able to measure eye deviation with your prism set. How can you estimate the amount of eye deviation?
You can do this by measuring the corneal light reflex in relation to the underlying pupil (Hirschburg test). For every mm of light deviation, you have approx 14 diopters of strabismus.

4. What's the difference between a tropia and a phoria? How can you differentiate this on exam?
This is exactly the kind of question you'll probably get asked during a pediatric or neuro clinic. A tropia is a deviation that is there ALL the time. A phoria is intermittent … tends to occur more with fatigue.

As for how to pick these up … it's hard to describe, so you should have one of the residents demonstrate how these are done:

Tropias: cover-uncover test

Phorias: cross-cover test

5. The actress Lucy Liu (from Charlie's Angels) brings in her new baby. The Asian American actress is concerned because Drew Barrymore said her baby's eyes looked "crooked." Casual inspection shows a healthy 3-month old baby who appears mildly esotrophic (cross-eyed). How would you measure ocular alignment in this child.

You can check the corneal light reflex with a penlight.

6. The corneal light reflex appears to be correctly centered (normal Hirschburg), yet the child still looks esotrophic. In fact, the child looks a lot like her attractive Asian mother. What's going on?
This sounds like pseudostrabismus from epicanthal folds. This nasal skin creates the illusion of crossed-eyes. As babies get older the nasal bridge becomes prominent, and this illusion usually goes away.

7. What retinal signs will you see in child abuse?
Retinal hemorrhages at all levels of the retinal in all four quadrants of the eye. Schisis cavities are relatively specific.

8. Parents say that a child fell from her crib and hit the floor. Do you think this would cause a fracture and the retinal findings of shaken baby?

Kids are relatively bouncy … but this story COULD account for the skull fracture. However, it takes a LOT of traumatic force to create large retinal hemorrhages -- this story sounds fishy!

9. You diagnose an infant with shaken baby syndrome. The mother says she has seen her four-year-old daughter shaking the infant. Is this possible?

A four-year old doesn't have the strength to pick up a baby and shake it. You need an adult or adult-size youth with strength enough to create that kind of damage. The violence needed is usually extreme and repetitive.

10. What other findings on exam/imaging might you see in a shaken child?

Intracranial hemorrhage and edema, skull-rib-long bone fractures. You might also find bruising on the body trunk and under the armpits. Supposedly, these kids can have a torn frenulum (the piece of tissue under the upper lip that connects the lip to the gumline).

11. A mother is concerned that rough play with her baby (tossing-in-the-air, bouncing the child on the knee, etc.) could cause shaken baby or brain damage. Can it?

Such playful practices don't cause brain injury.

12. Can seizures cause retinal hemorrhages similar to shaken baby? How about vaccinations?

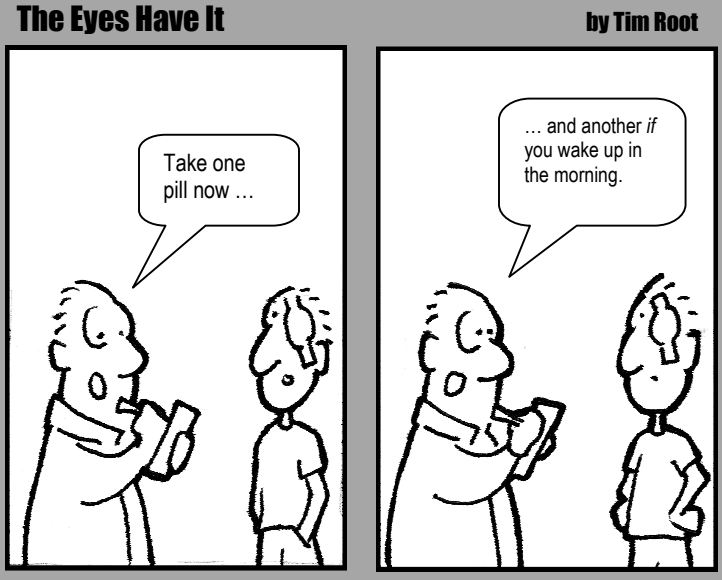
No.

13. Name three causes for a leukocoric pupil.

Cataract, retinoblastoma, and retinopathy of prematurity.

Chapter Nine

Eye Trauma



Trauma

by Tim Root
(last updated 6-17-06)

You probably won't see much trauma in our clinic unless you spend a night on-call with one of the first year residents (or rotate through any emergency room).

When I picked ophthalmology as a career, I never dreamed there would be so many midnight emergency room consults. Little did I know that so many people out there are punching each other in the face and slamming their heads into airbags. Old people seem to tip over face-first onto concrete surfaces and there are a ton of people out there welding, hammering, and "grinding metal" without proper eye-protection. Repeated exposure to all this eye trauma changes your world outlook - I now dread baseball season, Christmas BB guns, and fireworks. That being said, it's our duty to help these people no matter how odd their injury … that's what being a professional is all about.

Famous Quotes

I thoroughly disapprove of duels. If a man should challenge me, I would take him kindly and forgivingly by the hand and lead him to a quiet place and kill him.

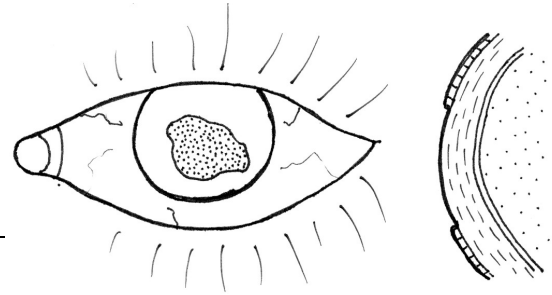
Mark Twain

Here are some of the common traumas you'll likely see coming through the ER.

Corneal Abrasions:

The surface of the eye is covered by a thin "skin" of epithelium. This "rug" of cells can be scraped off if the eye is scratched. These surface abrasions are the most common injuries to the eye and we see them daily.

There are more nerve-endings in the cornea than any other place in the body so these injuries "hurt like the dickens," with patients complaining of excruciating pain and intense photophobia.

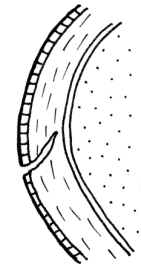


Fortunately, corneal abrasions heal very quickly

– sometimes within a day. You treat with aggressive lubrication and follow them daily until the epithelium heals over, to insure the raw wound doesn't become infected. Many will treat with empiric erythromycin as well, reserving more aggressive antibiotics like ciprofloxacin for contact lens wearers and "dirty wounds" caused by tree branches, etc.

Abrasions are easy to see, even without a microscope. The raw surface will uptake fleurosceine and glow bright green under a blue light. If an abrasion becomes infected, you'll see a white infiltrate at the wound – any abrasion with an infectious infiltrate is officially called a "corneal ulcer." Depending upon the size and location of an ulcer, you may need to culture the wound and tailor your antibiotic coverage accordingly.

Corneal Lacerations:



Most corneal scratches only involve the surface epithelium. If a cut goes deeper into the stroma, then you have a laceration.

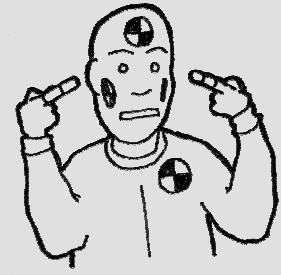
With any laceration you want to insure that the cornea hasn't been perforated. You can check corneal integrity with the "seidel test." You place a strip of fleuorosceine paper over wound and see if dye flows down (indicating leaking aquous fluid).

If a patient is "seidel positive" than you have an open-globe injury … time to call in your chief resident and attending for surgical repair!

Orbital Wall Fractures:

The bony orbital walls are thin and tend to break with blunt globe injuries, especially the orbital floor and medial walls. These orbital fractures are very common and I see them on a weekly basis, usually at 2 in the morning.

Tim's Bumper-Face Theory



The face is designed like a crash-tested car … with many areas that are designed to crumple and diffuse energy upon violent impact. The sinuses are air-filled crumple zones that protect the brain and other vital structures. When the eye is hit, orbital contents (usually fat) often herniate into one of the sinuses … this is a good system, as it keeps the eye from exploding from high pressure of an impact.

Floor fractures are very common … and can be a major headache for the first year resident as they tend to occur at 2am in the morning. Most of these patients heal up well within a few weeks, with antibiotics and "no nose blowing." However, if an ocular muscle becomes entrapped in the "trap door" they may require surgery.

Most of the time these orbital bones heal fine with no long-term problems, with patients merely having a great deal of orbital and periorbital swelling which resolves over the next few weeks.

However, sometimes the break will create a “hinge” or “trapdoor” which entraps fat or extraocular muscles. If there is significant entrapment or enophthalmos, we need to repair the break. In surgery we can release the muscle and place a plate of material over break to keep orbital contents from herniating back through the defect.

When evaluating orbital fractures, focus on the following things:

1. Vision, color: Make sure the optic nerve isn’t involved.

2. Extraocular movements: Usually decreased from swelling, but make sure there isn’t any gross muscle entrapment.

3. Proptosis: Measure the degree of proptosis or enophthalmos using the Hertel ruler.

4. Palpate: Feel along the orbital rim for step-off fractures and emphysema air.

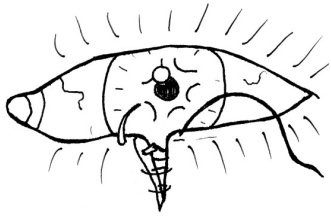
5. Sensation: Check sensation of the V1 and V2 sensation on the face.

Most of these patients do fine and we see them a week later. In the meantime, I’ll treat empirically with Keflex or Augmentin, advise Afrin nasal spray, and recommend “no nose blowing” (you don’t want to blow air from the sinuses into the orbit).

Lid Lacerations:

When evaluating the lids, you need to determine a few things … does the laceration involve the lid margin, full thickness, and how close is it to the canalicular (tear drainage) system.

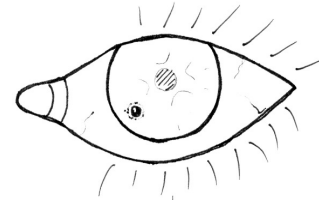
Most lid lacerations are easy to repair, though special effort is made to align the lid margins … you don’t want someone to have a notch in their eyelid or have eyelashes disaligned.



If the laceration is medial (near the nose) you need to worry about the canaliculus … repair of this drain is much more involved.

Metal on Metal:

Small pieces of metal often fly into the eye … either by scrapping metal, during welding, or hammering. Despite evidence to the contrary, these patients are always emphatic that they were wearing eye protection.



Particles of metal easily stick onto the cornea … causing a small abrasion and discomfort. Metal rusts quickly and will form a rust ring around the object within a day. You can remove metal objects and rust rings at the slit-lamp using a 25 guage needle. You can also use a small hand-held dremel-drill to burr off the rust-ring, though sometimes you'll have to leave some of the ring there.

Anytime you have metal-striking-metal injuries, you have to entertain the possibility of an intraocular foreign body. Small metal fragments can hit the eye at high speed and leave little or no signs of entry. Metal is very toxic to the retina and can kill the retina if not detected. I get either a thin-slice CT scan and/or a PA and lateral x-ray of the head (cheaper) to look for metal pieces not seen on exam. You want to avoid MRI … no point in creating a moving projectile inside the eye.

Chemical Injuries:

Household cleaners contain toxic substances like bleach or ammonia that are extremely toxic to the eye. The first thing you do with any chemical injury is:

… Irrigate, Irrigate, Irrigate, Irrigate, Irrigate, Irrigate, Irrigate …

The final visual outcome for a chemical burn is going to depend upon how quickly the chemical is washed out of the eye. If a patient calls you with a chemical conjunctivitis, tell them to immediately wash their eyes out! If the ER calls you with a chemical conjunctivitis, tell them to start irrigating immediately … several liters in each eye. Then grab your equipment and pH paper and head on down there!

Acids are less dangerous than bases as acids tend to precipitate denatured proteins that limit tissue damage. Bases just keep on going like the Energizer Bunny so you need to continually irrigate and check the pH until normal.

On exam you want to check the state of the cornea. A red, injected conjunctiva is actually a good finding … if the conjunctiva is white, that means its blanched out from damage. Be sure to flip the lids and irrigate/sweep the fornices to remove any material that may be retaining chemicals.

Chemical injuries can lead to significant scarring that may require corneal transplant … so you want to be very aggressive with that irrigation!! The emergency room has access to a simple device called a Morgan lens to help irrigate. As you can imagine, little kids hate this thing and have to be restrained when using it.

Fun Fact!

Speaking of abrasives … early Romans used human urine as a mouthwash to brush their teeth. The ammonia has strong cleaning powers. In fact, urine was an important component of toothpaste well into the 1700's. Lesson: If you can avoid it - don't get urine in your eyes.

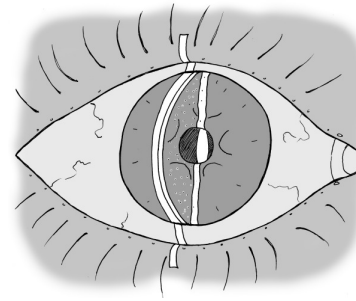
Traumatic Iritis:

The anterior chamber can become inflamed after blunt trauma. Patients will complain of painful sensitivity to light secondary to iris/ciliary spasm. Fortunately, traumatic iritis generally runs a benign course with resolution of symptoms within a week.

On exam you look for "cells and flare" in the anterior chamber. Cells are inflammatory cells and iris pigment floating within the aqueous, while "flare" is diffuse protein that escapes through inflamed blood-vessel walls.

Individual cells can be difficult to detect at the slit lamp … and it doesn't help when the photophobic patient is squeezing their eyes shut and yelling at you. You'll find it helpful to turn the lights completely off and to make your light narrow, bright, and at an angle (like in this picture).

We treat these patients with steroids (assuming there isn't a corneal abrasion) to decrease the inflammation, cycloplegics to dilate the eye and decrease photophobia, and treat pressure if elevated. I generally use a medium-duration dilator like Cyclogyl several times a day … a dilating drop makes the patient feel better and forces the iris to dilate several times a day, keeping the inflamed iris from sticking to the underlying lens.



Hyphema:

A hyphema describes blood floating in the anterior chamber, which occurs after blunt eye trauma. If the bleed is large, the blood will settle to the bottom of the AC and appear "layered." If the entire AC is filled with blood, you'll get an "8-ball hyphema." Most of the time, however, the bleeding is microscopic and can only be seen as "red cells" floating in the aqueous fluid.

Blood typically clears up well … though you can get staining of the cornea if the patient also has coexisting high pressure. We recommend that patients sleep with their head elevated (to help the blood settle) and to avoid straining. We'll typically give steroids (to decrease the inflammatory response) and a cycloplegic dilating drop to help with photophobia … this

dilation also keeps the iris from sticking to the underlying lens and forming synechia.

A couple of key points with hyphemas:

1. With African Americans, consider checking for sickle cell disease. If they do have sickle cell, avoid carbonic anhydrase inhibitors as they cause a relative acidotic state that worsens sickling.

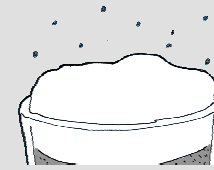
2. Follow these patients daily, as the bleeding can get worse. The main danger time is days 3 to 5 … this is when clots can contract and rebleed. You need to watch their pressure as the blood can clog-up the trabecular meshwork.

3. After the blood has completely resolved, perform a thorough goinioscopy exam to access for "angle recession." This is when the ciliary body splits from the blunt trauma -- this is a sign (but not a causative factor) that the patient may eventually develop glaucoma in that eye down the road.

Speaking of fluid layering …

Fun Fact!

The "black and tan" tradition of beer mixing originated over a thousand years ago when Viking explorers raided the Celtic islands. The Vikings would mix their lighter northern beer with the local dark beers. Later, the term "black and tan" came in use to describe the uniforms worn by cruel British solders sent to Ireland in the early 1920's to suppress uprisings.



A black and tan is most commonly constructed with Bass Ale (an English bitter) and Guinness (an Irish Dry Stout). The Guinness is pored over the lighter colored beer using an inverted spoon to disperse the Guinness and decrease mixing. The beers have different densities and so will remain "layered."

The "black and tan" is enjoyed by beer enthusiasts who find a straight stout too harsh. However, you may want to avoid ordering one in Ireland because of its historical relevance. And of course … Guinness tastes better unadulterated!

Open Globe Injuries:

The eye can be perforated many ways … over the last six months I've seen firecracker explosions, several MVAs, and domestic accidents that have destroyed the eye. Visual outcome is usually terrible and the eye will often need later enucleation.

Open globe injuries need to be evaluated in the operating room. One thing to remember - if you suspect an open globe injury, cover the eye with a shield and don't push on it. You could extrude the eye contents (pop it like a grape) if you push on the eye.

1. You have a contact lens wearer with a small corneal abrasion. He is in excruciating pain and requests his eye be pressure-patched for comfort. Will this speed up healing?

Patching may speed healing by keeping the eye immobile and lubricated … but you should never patch an abrasion that might fester an infection. Thus, you don't patch contact lens wearers - you don't want a pseudomonas infection brewing under that patch!

2. What's the easiest way to see a corneal abrasion? How often do you need to follow simple, non-infected abrasions?

Abrasions are easiest seen under fleurosceine in the slit-lamp. I can sometimes diagnose the abrasion with a simple penlight … the edges of the abrasion creates a circular shadow on the iris underneath. You'll want to measure the epithelial defect and see them daily until it heals to make sure they don't become infected.

3. What is the Seidel test?

This is a check to see if a corneal laceration has perforated through the cornea into the anterior chamber. Basically, you're using fleuoresceine to look for leaking aqueous.

4. What findings would prompt you to take a patient with orbital floor fracture to surgery?

If the patient had muscle entrapment or significant enophthalmos. Most patients have some degree of EOM restriction from soft-tissue swelling. Entrapment causing reflexive bradycardia would also push you toward surgery.

5. What portion of the eyelid do you worry about with lid lacerations?

If the laceration is medial (near the nose) it could involve the tear drainage pathway, and this repair is more complicated.

6. A patient accidentially splashes a large amount of bleach-based cleaner in her eye. What should she do?

Wash it out immediately … the faster, the better!!!! If an ambulance picks her up, have them irrigate in route, and alert the ER to irrigate her eyes as soon as she hits the door.

7. What is the best way to test the pressure in an eye with a likely open-globe … with slit-lamp applanation or with the hand-held tonopen?

If you suspect open globe, you don't want to be mashing on the eye … so neither of these is correct. Trick question … hahahahaha!

8. How often should a patient with a hyphema be seen and why?
These patients need to be seen daily to check pressure. Also, the highest risk for re-bleed is post-trauma day 3 – 5, as this is when clots begin to retract.

9. An African American presents with hyphema after trauma. What additional workup might you consider? Are there any medications you would avoid?

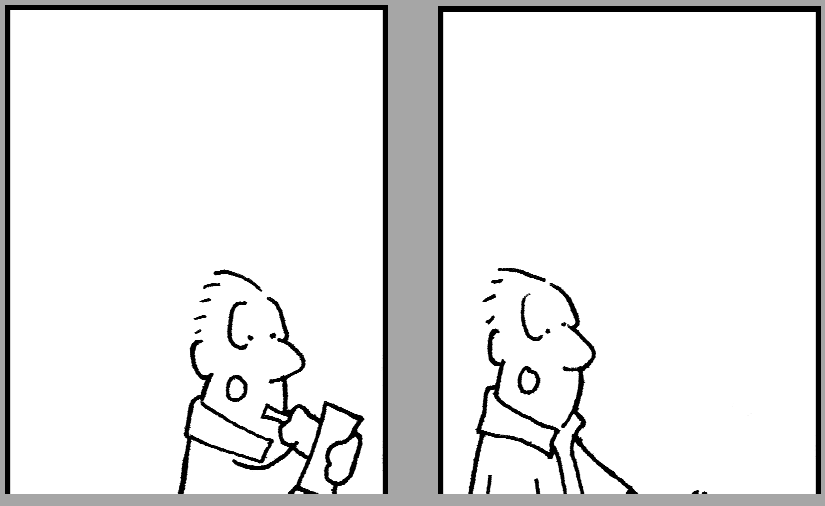
You may consider getting basic coagulation labs and a sickle prep. Avoid CAIs as these promote acidosis and thus worsen sickling.

10. What two beers are most commonly used when making a "black and tan." Which beer goes on top?

A black-and-tan is made with Bass Ale and Guinness Stout … the Guinness goes on top and is usually pored over a spoon to keep it from mixing.

Chapter Ten

Drugs & Toxicity



Drugs and Toxicities

by Tim Root
(last updated 7-4-06)

Pharmacology can be a painful subject to study … it's hard to memorize these medications unless you prescribe them on a daily basis. It's even harder for the medical student because they don't usually write out the actual prescription pad. Ophthalmology has it's own collection of favorite medications and eye-drops, that are much different than general medicine … so I'm going to simplify this chapter as much as possible. Here are the most common medications we prescribe and some important facts about each:

Antibiotics:

In our clinic, we prescribe topical antibiotics for bacterial conjunctivitis and corneal ulcers. We typically give either:

a. **Erythromycin:** The most prescribed medication we use. Good for just about anything with very few allergic reactions. It's cheap so we never have any samples.

b. **Ciloxan (ciprofloxacin):** We often use this fluoroquinilone because it covers pseudomonas. We use this ointment with any bad-looking conjunctivitis or a corneal ulcer (especially in a contact lens wearer or dirty wound). The only thing with cipro is that it doesn't cover certain strains of streptococcus. If you've actually cultured streptococcus (or have an ulcer that isn't improving) you should switch to a combination of fortified (mixed special at high concentrations by pharmacy) antibiotic.

c. Vigamox (moxifloxacin): An expensive 4th generation fluoroquinilone with super ocular-penetrance that we use after surgery. It doesn't cover pseudomonas, though.

To cover skin-flora infections, such as preseptal cellulites, dacryocystitis, and orbital wall fractures (thus a connection between sinuses and orbit) we give the standard cellulutis/sinusitis medications used in other specialties. These are (in order of power and cost):

a. **Augmentin:** Kicks butt, very expensive, and can give diarrhea. This is a third-generation aminopenicillin that also covers gram negatives. This broader spectrum is great for children.

b. **Keflex (cephalexin):** Pretty good (I usually recommend this for wall fractures). This is a first-generation cephalosporin with broad gram-positive coverage. This is good for adults (who are more likely to have gram positve infection) and relatively inexpensive. The other surgical services prescribe this stuff like candy.

c. **Dicloxacillin:** Not very good, but dirt-cheap. I'll give this if the patient can't afford Augmentin or Keflex. It's better than nothing.

We also use a lot of **Tobradex** ointment (combination of tobramycin and a steroid) after surgical procedures as it combines antimicrobial and anti-inflammatory properties in one cheap salve.

A "controversial" note on antibiotic resistance (personal opinion)

One fear in general medicine is that over-treating minor bacterial infections with long-term low-dose antibiotics will create resistant bugs. This potential problem is less relevant to ophthalmologists. This is because MICs (minimal inhibitory concentration) for antibiotics are based on *blood* level concentrations of antibiotics. We usually apply the antibiotic directly to surface infections, so we are slamming the bacteria with antibiotic concentrations orders of magnitude higher than normal… the bacteria doesn't have time to develop the massive resistance needed for all that antibiotic.

We still culture corneal ulcers to identify organisms and tailor coverage (bacterial, pseudomonas, fungal, etc.) but sensitivities are generally less important for us for this reason.

Dilating the Pupil:

To dilate the eye (mydriasis) we need to "stimulate the pupil." For adult patients we use two medications – phenylephrine 2.5% and tropicamide. Here's a rundown of the various dilating drops and their indications.

a. **phenylephrine** – a sympathetic stimulator. This drop will also constrict conjunctival blood vessels and "get the red out." This vessel blanching also has some diagnostic utility in determining what level of blood vessels in the conjunctiva/episclera/sclera is actually injected.

- -

b. **tropicamide (a.k.a. Mydriacyl)** – A short-acting parasympathetic blocker. Remember, these parasympathetic antagonists also paralyze the ciliary body (which controls the lens shape) and make it hard for the patient to read.

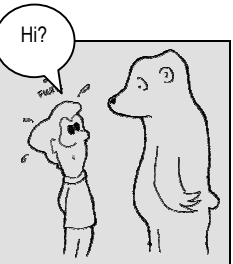
c. **cyclopentolate (a.k.a. Cyclogyl)** – A medium-acting parasympathetic blocker. We might use this in a child because tropicamide sometimes isn't strong enough. We also give this to people with "photophobia." By paralyzing the ciliary body, the iris/ciliary body doesn't spasm in bright light, thus making the photophobic patient more comfortable.

d. **atropine** – A long-acting parasympathetic blocker. We'll use this to help with photophobia. Also, atropine seems to have some anti-inflammatory properties of its own. Don't give this drop by

mistake, though, or you'll mess up someone's reading ability for a week!

Mnemonic

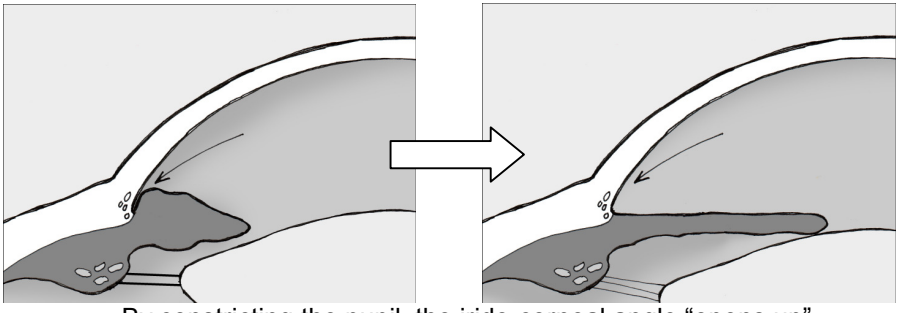
Remember … if you see a bear in the woods, your sympathetic "fight or flight" response kicks in. Your eyes will **dilate** so you can see better as you run for your life! In other words: sympathetics make your eyes dilate, parasympathetics constrict.



For dilating adults we typically use phenylephrine and tropicamide and you'll find these two drops in every room. Don't dilate the eyes if there is any question of an APD … go find a resident. If you're in neuro clinic, the attending will probably want a pupil check as well.

Constricting the Pupil:

Pilocarpine will constrict the pupils and cause "myosis." Pilocarpine used to be a common drug used to control pressure as it acts directly at the trabecular meshwork to increase outflow. These days, however, we primarily use it to constrict the pupil before LPI (laser peripheral iridotomy) … as it's easier to burn through a thin, flat iris. We can also use pilocarpine in cases of acute glaucoma, as constriction will open up the drainage angle as in this picture:



By constricting the pupil, the irido-corneal angle "opens up"

Decreasing Pressure

To decrease the pressure in the eye you need to either

1. Decrease the amount of aqueous fluid produced.
2. Increase the outflow of aqueous from the eye.

Topical **beta-blockers** like timolol work by decreasing aqueous humor production at the ciliary body. Unfortunately, systemic side effects can occur

from nasal absorption, making it especially important to ask your patients about history of asthma, COPD, and cardiac problems.

Alphagan is an **alpha-agonist** that works by both decreasing aqueous production and increasing uveo-scleral outflow. Alphagan can cause some allergy problems and we don't use it in infants as it can cause CNS depression.

Prostaglandin analogues like latanoprost (Xalatan™) or travaprost (Travatan™) are the newest of these glaucoma drugs and are often our first line medications as you only need to use them once a day. Prostaglandins increase aqueous outflow via the uveal-scleral tract. These drugs do have some side effects, though, as they can make eyelashes grow thicker (many patients actually like this), and in a few patients may darken the iris color, turning green and blue eyes brown. The prostaglandins can also irritate the eye, so we prescribe them to be taken right before bedtime … probably so that people are sleeping and don't notice this irritation.

Steroids:

We use steroid eyedrops in many patients because they decrease inflammation. You'll see drops like Pred Forte (prednisolone acetate) used postoperatively. We also use these steroids in cases of traumatic iritis, uveitis, and hyphemas to decrease the inflammatory response in the anterior chamber. For longer or continuous duration, we can inject a Kenelog (triamcinalone) steroid depot underneath the conjunctiva and for some retinal conditions we inject it directly into the vitreous.

While steroids work great, we typically avoid them in cases of epithelial abrasions as they may delay healing and increase risk of bacterial and viral infection. We never use in cases of herpetic ulcers! One other side effect you should know about: some patients are "steroid responders" who develop high intraocular pressures with their steroid eyedrops. This typically takes weeks of steroid use to occur. Also, long-term steroids can create cataracts.

Patanol and allergies

Allergy season starts in February and patients flock in droves to the on-call clinic complaining of bilateral eye swelling, irritation, and itching. Conservative treatment focuses on eyedrops and cold compresses. We also prescribe Patanol … currently, the number one allergy drop that we use. A second-generation anti-allergen, it is selective for H1 receptors and has less cholinergic side effects like dryness and drowsiness – basically, it's like Claritin for the eye.

Of note: These second generation allergics have less drug-drug interactions. Some of the first generation medications like Seldane, which was pulled from the market, would combine with erythromycin and cause cardiac problems and death.

Drug Toxicities

Preservatives

Many eyedrops contain preservative chemicals to keep them from growing bacteria. This preservative is also toxic to the eye, which becomes a problem when patients are taking many drops throughout the day. The biggest preservative problem is BAK (benzalkonium chloride), a drop preservative found in most of the glaucoma drops.

Many lubricating eyedrops have a preservative as well … you can give the single-dose preservative-free drops, but they cost more. I like to recommend Refresh and Gentel brands as the peroxide-based preservative breaks down into harmless oxygen and water and can be used more frequently. Ointments don't need preservatives as bacteria need an aqueous environment to flourish.

Anesthetic Eye Drops

For topical anesthesia we use proparacaine, while the emergency room uses tetracaine (which stings more). If you suspect a herpetic infection, be sure to check for corneal sensation prior to using that anesthetic! Also, never prescribe an anesthetic to a patient … as it will kill off the cornea. Don't do it! Desperate patients have been known to steal bottles, so be conscious of where you place them.

Plaquenil

Originally used as an anti-malarial medication, plaquinil is often prescribed in the rheumatology clinic to relieve inflammation of rheumatoid arthritis and other autoimmune diseases. However, the drug can cause retinal toxicity so all these arthritic patients need to be seen by ophthalmology for a baseline exam and periodic checkups. Ocular side-effects are actually quite rare and doctors in Europe don't even bother with these checkups. Our society is more litigious, though, so we're stuck seeing them.

Affected patients develop blind spots in the central or para-central vision, and complain of other visual symptoms like flashing lights, photophobia, and night blindness. The classic retinal finding is the "bull's eye maculopathy" with the disease forming a ring around the macula. Red vision change is often the first visual symptom so we monitor using a red visual field test.

Metal Toxicities

Metal is not good for the eye … and metal foreign bodies in the vitreous chamber can kill off the retina. Sometimes, systemic metal toxicities can be seen in the eye especially at the clear cornea.

Wilson's disease is a hereditary disorder seen in young people where the body can't excrete copper in the bile. Copper buildup is bad and patients have deadly CNS and liver problems. Lab work shows high liver and urinary

copper, and a low ceruloplasmin (the blood protein that carries copper). Copper deposition can show up in the eye as a Kayser-Fleisher ring in the cornea. This green or rust-brown ring is difficult to see, and generally needs to be viewed at the slit-lamp using gonioscopy.

The poor metals (like iron and calcium) have to sleep up in the attic (up in Bowmans layer) while the rich metals (like copper, silver, and gold) get to stay comfortable down below in Descemets.

1. What's the most common topical antibiotic we use in our clinic? What antibiotic do we use for pseudomonas coverage?

Erythromycin ointment is our mainstay antibiotic. Ciprofloxacin covers pseudomonas.

2. Will a sympathomimetic drug dilate or constrict the pupil?

Sympathetics, like the common dilator phenylephrine, dilate the eye. Remember … with the fight-or-flight reflex the pupils get big.

3. Many over-the-counter cold medications caution use in patients with glaucoma. Any idea why? What should you tell your open-angle glaucoma about these medications?

Many cold-medications and antihistamines have sympathetic stimulating medications to constrict nasal blood vessels. Sympathetics will also dilate the eyes … if a patient has shallow or "occludable angles," then a bunched-up iris could narrow their drainage angle even more and cause an acute glaucoma. When we see patients in clinic, we always check the anterior-chamber depth to make sure they won't occlude when we dilate them. Patients with deep "open angle glaucoma" are not at risk for sudden angle-occlusion, and can take these medications safely.

4. You see a patient with traumatic iritis (painful inflammation of the anterior chamber) complaining of pain and photophobia. What can you give to decrease the inflammation? What can you give this patient to make them more comfortable?

Topical steroid drops like Pred Forte will help with the inflammation. This patient is suffering from photophobia (pain with bright lights) and could benefit from cycloplegia … the parasympathetic blockers like Cyclogyl or atropine will paralyze the ciliary body, dilate the iris, and make the patient feel better. Also, by keeping the inflamed iris "moving" with dilation, you keep it from sticking to the lens underneath.

5. A young businesswoman comes in for an eye exam. The techs inform you that they've run out of tropicamide (mydriacyl) but still have atropine on stock for dilating. What should you do?

Well … don't use atropine for routine dilation! You'll cycloplege (paralyze the ciliary body muscle) for a week or more and this young professional won't be able to read during this time.

6. A man presents as followup from the emergency room for a small corneal ulcer. Despite the sample bottles of ciprofloxacin and tetracaine he was given in the ER, his eyes seem to be getting worse. What could be going wrong?

The problem is the tetracaine … NEVER give your patients anesthetic drops … they are toxic to the cornea and will kill the eye. This patient could develop corneal scaring and end up needing a transplant. This has happened to patients in the past if they steal anesthetic drops from clinic or are erronously prescribed in ERs. Personally, I think non-ophthalmologists should only prescribe three medications for the eye: erythromycin, ciprofloxacin, and possibly Patanol for allergies. This is a personal opinion, though, and may change with experience.

7. You are consulted for a patient in the MICU. The young patient is on a ventilator with cirrhosis, mental changes, and a low ceruloplasm level. The medicine team wants you to look at her eyes … why?

Sounds like the team wants you to look for signs of a Kayser-Fleisher ring to confirm Wilson's Disease. Unfortunately, you probably won't be able to see much … you need a careful goniocopic exam at the slit-lamp to see one as copper deposits in the deep Decemet's membrane (along with the other "rich metals" like gold and silver).

8. An infant with congenital glaucoma is being treated with timolol, alphagan, and xalatan. The mother complains that her child seems sleepy and lethargic. What could be going on?

The alpha-agonist could be causing this. We don't use alphagan in infants as it causes CNS depression.

9. Are there any eyedrops that can negatively affect a fetus?

I am not aware of any eyedrops have ever been proven to have teratogenic effects. There are many systemic medications that we prescribe, though, that do … such as doxycycline for the treatment of blepharitis.

The

Appendix!

Ophthalmology Abbreviations

A or Acc	accommodation
AC	anterior chamber
AC/A	accommodative convergence/accommodation ratio
ALT	argon laser trabeculoplasty
APD	afferent pupil defect
ARMD	age-related macular degeneration
ASC	anterior subcapsular cataract
BLP	bare light perception
BRAO	branch retinal artery occlusion
BRVO	branch retinal vein occlusion
CE	cataract extraction
CF	confrontational fields or count fingers
CL	clear, contact lens
CRAO	central retinal artery occlusion
CRVO	central retinal vein occlusion
CS	conjunctiva and sclera
CSME	clinically significant macular edema
CSR	central serous retinopathy
D	diopters
DQ	deep and quite
DR	diabetic retinopathy
EOG	electro-oculogram
EOM	extraocular movements
ERG	electroretinogram
ERM	epiretinal membrane
ET	esotropia
EXT	extremities
FA	fluorescein angiography
FLK	funny looking kid (don't use)
FOL	follicles
HE	hard exudates
HM	hand movement
HSV	herpes simplex virus
HT	hypertropia
I	iris
IMHO	in my humble opinion
IOP	intraocular pressure
J1,J2…	Jaeger (near vision scale J1+=20/20)
K	cornea
KCN	keratoconus
KPs	keratic precipitates
L	lens
LASIK	laser in situ keratomileusis
LOL	laugh out loud
LP	light perception
LPI	laser peripheral iridotomy
LL	lids and lacrimation
LLL	left lower lid
LUL	left upper lid
MA	microaneurysms
MP	membrane peel
NLP	no light perception
NPDR	non-proliferative diabetic retinopathy
NSC	nuclear sclerotic cataract
NV	neovascularization
NVA	neovascularization of the angle
NVD	neovascularization of the disk
NVE	neovascularization elsewhere
NVG	neovascularization glaucoma
NVI	neovascularization of iris
OD	oculus dexter (the right eye)
OS	oculus sinister (the left eye)
OU	oculus uterque (both eyes)
P	pupils
Pap	papillae
PAS	peripheral anterior synechiae
PCO	posterior capsular opacification
PD	pupillary distance
PDR	proliferative diabetic retinopathy
PED	pigment epithelial detachment
PED	persistent epithelial defect
PEE	punctate epithelial erosion
PH	pinhole
PHNI	pinhole no improvement
PHPV	persistent hyperplastic of primary vitreous
PI	peripheral iridotomy
PK	Penetrating keratoplasty (corneal xpl)
POAG	primary open angle glaucoma
PPV	pars plana vitrectomy
PRP	panretinal photocoagulation
PSC	posterior subcapsular cataract
PVD	posterior vitreous detachment
PVR	proliferative vitreoretinopathy
PXS	pseudoexfoliation syndrome
RAPD	relative afferent papillary defect

RD	retinal detachment
ROFL	roll on floor laughing
RLL	right lower lid
ROP	retinopathy of prematurity
RP	retinitis pigmentosa
RPE	retinal pigment epithelium
RUL	rright upper lid
Sc	sans correction (no glasses)
SLE	slit-lamp exam
SLK	superior limbic keratoconjunctivitis
SRF	subretinal fluid
Sph	spherical lens
T	pressure
Tap	pressure (applination)
Tono	pressure (tonopen)
V or Va	vision or visual acuity
Vcc	vision (with correction)
VEP	visual evoked potential
Vsc	vision (sans correction)
VH	vitreous hemorrhage
Vit	vitreous
W&Q	white and quite
WNL	within normal limits
XOXO	hugs and kisses
XT	exotropia

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